COMPLICATIONS OF REGIONAL ANESTHESIA

Etiology - Signs and Symptoms - Treatment

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By

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DEDICATION

To our children Barbara, Nancy, Daniel and Susan, who have enriched our lives with their antics, problems, happiness and love

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"It is unfortunate, considering that enthusiasm moves the world, that so few enthusiasts can be trusted to speak the truth"

A J Balfour

Introduction

In 1952, Ralph Knight, then *Professor of Anesthesiology* at the University of Minnesota School of Medicine invited me to participate in a continuation course in anesthesiology for the general practitioner One of my assigned subjects was titled "Complications of Regional Anesthesia and Their Treatment" Following the presentation Frederick H Van Bergen who at the present time is Associate Professor and Director, Department of Anesthesiology, at that institution suggested that this material be expanded and set forth in a book

Since that time the literature on the complications of regional block procedures has been reviewed and our own experiences with complications from these techniques analyzed It was found that the complications of regional blocks are primarily due either to the untoward results of the normal pharmacological actions of the blocking solutions used or to trauma associated with the technique They may be either catastrophic, i e, occur in 1 to 30 minutes following regional block or take days or weeks to develop

The untoward results of the normal pharmacologic actions of a drug usually result in the catastrophic type of complication which is reversible in most instances as the drug's action is dissipated provided that immediate and correct treatment is given If rapid diagnosis and correct therapy are not instituted then the severe hypotension oxygen want, unconsciousness and/or vomiting—signs and symptoms which reflect altered body physiology—result in irreparable tissue damage Haldane emphasized this point "Anoxemia not only *stops* the machine but wrecks the machinery!" On the other hand trauma to the tissues usually produces slowly developing pathological changes which while they may result in serious sequelae, do not immediately threaten the patient's life Obviously these slowly developing sequelae should be treated correctly but time alone will reveal the outcome *Every physician or dentist using a regional block technique is obligated to be fully aware of the inherent dangers of either type of complication If he does not have the knowledge and facilities to institute effective treatment of these complications he should not employ the technique!*

This book represents an attempt to present the complications of regional anesthesia to the profession in a concise complete and explicit fashion which will be of *practical use* in an everyday practice An effort has been made to make each chapter in this book complete within itself so that the physician will not be burdened by having to read the whole book in order to review *one* complication This necessitated some repetition which it is hoped will not be too distasteful to the majority of readers

The material in this book is divided into three parts Part I considers the complications of local infiltration and peripheral nerve block, Part II reviews the complications of spinal (subarachnoid) and epidural (peridural) block and Part III discusses incidental complications

Although this book is entirely devoted to the complications of regional block procedures, this should not be construed to mean that complications following regional nerve blocking occur frequently or that this type of anesthesia is more dangerous than other types

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The untoward results of the normal pharmacologic actions of a drug usually result in the catastrophic type of complication, which is reversible in most instances as the drug's action is dissipated, provided that immediate and correct treatment is given. If rapid diagnosis and correct therapy are not instituted, then the severe hypotension, oxygen want, unconsciousness and/or vomiting—signs and symptoms which reflect altered body physiology—result in irreparable tissue damage. Haldane emphasized this point: "Anoxemia not only stops the machine but wrecks the machinery!" On the other hand, trauma to the tissues usually produces slowly developing pathological changes which, while they may result in serious sequelae, do not immediately threaten the patient's life. Obviously, these slowly developing sequelae should be treated correctly, but time alone will reveal the outcome. *Every physician or dentist using a regional block technique is obligated to be fully aware of the inherent dangers of either type of complication. If he does not have the knowledge and facilities to institute effective treatment of these complications he should not employ the technique!*

This book represents an attempt to present the complications of regional anesthesia to the profession in a concise, complete and explicit fashion which will be of practical use in an everyday practice. An effort has been made to make each chapter in this book complete within itself so that the physician will not be burdened by having to read the whole book in order to review one complication. This necessitated some repetition which it is hoped will not be too distasteful to the majority of readers.

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PART I

COMPLICATIONS OF LOCAL INFILTRATION AND PERIPHERAL NERVE BLOCK

PART I

COMPLICATIONS OF LOCAL INFILTRATION AND PERIPHERAL NERVE BLOCK

Introduction to Part I

THE COMPLICATIONS considered in this part of the book are those which are primarily encountered following local infiltration and peripheral nerve block. Fortunately, these procedures if carefully executed seldom are followed by the serious complications—namely, systemic toxic reactions, an inadvertent total spinal block or a bilateral pneumothorax, sequelae which may result in cardiac failure and death in 1 to 30 minutes. Because of their catastrophic nature these complications are considered first, although they may not occur as frequently as the less serious ones such as tissue swelling, overflow onto nerves not intended to be blocked or bleeding into tissues.

It must be remembered that if the epidural (peridural) or subarachnoid spaces are entered while executing a peripheral nerve block all complications found in Part II may develop and that those complications which are incidental to regional block procedures and discussed in Part III may occur.

whether due to the local anesthetic agent or the vasoconstrictor drug should not be viewed lightly since they may herald the onset of a more serious situation progressing to convulsions, respiratory or cardiac failure, and death

The seriousness of overlooking a systemic reaction from a local anesthetic drug or misjudging its magnitude can be vouched for by the following reports In 1924 and again in 1928 the reports of a committee of the American Medical Association which studied reactions from local anesthetic agents reviewed 57 deaths from various local anesthetic drugs[9,10] The greater number of these deaths followed topical applications In 1951, Ireland *et al*[11] after a survey of the largest otolaryngological clinics in the United States and Canada, reported 7 fatalities due to either cocaine hydrochloride or Pontocaine (tetracaine) hydrochloride in 39 278 instances of local anesthesia Furstenberg[12] reported 3 fatalities in 30 000 tonsillectomies using cocaine hydrochloride and Williams[13] 2 deaths in 6 378 nose-throat operations employing the same drug Schindler[14] found 3 fatalities from cocaine hydrochloride and tetracaine hydrochloride in a total of 22 351 patients undergoing gastroscopic examinations in 60 clinics in the United States, England and Australia While these studies point out the potential hazards of local anesthetic agents they also show their relative safety

Clarification and Definition of Terms —As noted previously systemic toxic reactions to local anesthetic agents reflect either an *allergy* or a *high blood level* of the drug and some clarification and definition of terms is needed so that those used in this book are clearly understood (see Table II page 8)

The word *allergy* is used by Dorland[15] and Vaughan and Black[16] as a general all inclusive term embracing anaphylaxis idiosyncrasy susceptibility hypersensitivity etc It covers the whole field of antigen antibody type reactions It should be noted that while in most instances Vaughan and Black[16] restrict the word anaphylaxis to experimental animal work they do feel it can be used in clinical allergy to denote shock and the term "clinical anaphylactic shock" is so used in this book to differentiate the true allergic shock from the severe cardiovascular respiratory collapse which occurs as a result of a high blood level (overdosage) of the drug From this discussion as well as from the definitions in Table II, it is easy to see why these various terms (idiosyncrasy, etc) have been applied by different authors to any systemic toxic reaction following the use of local anesthetic

TABLE I

CLASSIFICATION OF SYSTEMIC TOXIC REACTIONS

(Modified from Sadove et al and Collins)

CENTRAL NERVOUS SYSTEM EFFECTS

1 Stimulation of
   - a Cerebral cortex → convulsions
   - b Medulla
     - (1) cardiovascular center → increased blood pressure and pulse
     - (2) respiratory center → increased respiratory rate and/or variations in rhythm
     - (3) vomiting center → nausea and/or vomiting

2 Depression of
   - a Cerebral cortex → unconsciousness
   - b Medulla
     - (1) vasomotor → fall in blood pressure and rapid or absent pulse (syncope)
     - (2) respiratory → variations in respirations and/or apnea

PERIPHERAL EFFECTS

1 Cardiovascular (syncope)
   - a Heart → bradycardia, i e depression from Novocain etc or → tachycardia i e stimulation, from cocaine
   - b Blood vessels → vasodilatation

ALLERGIC RESPONSES

1 Skin → dermatitis etc

2 Respiration, 3 Circulation } → depression (clinical anaphylactic shock)

MISCELLANEOUS REACTIONS

1 Psychogenic

2 To other drugs e g vasopressors etc

CHAPTER 1

Systemic Reactions to High Blood Levels of Local Anesthetic Drugs

Two types of toxic reactions to drugs are usually recognized local and systemic[1] *Local* toxicity is defined as a reaction of the tissue at the site of the injection and is discussed in Chapter 10 page 93

Systemic toxic reactions are defined as those reactions which occur when absorption of a given drug takes place Systemic toxic reactions which occur following the injection of a local anesthetic drug are due to either a high blood level of the drug or a true allergy to it In most instances—98% or more—a systemic reaction indicates an overdose i e a high blood level of the drug for that particular individual—not a true allergy Such toxic reactions are seen following the use of large quantities of local anesthetic drugs in the following techniques local infiltration peripheral nerve block and occasionally in epidural blocks Systemic toxic reactions are seldom seen when small quantities of the local anesthetic drugs are used in methods such as spinal (subarachnoid) block

Classification of Systemic Reactions—Systemic reactions have been classified both according to their clinical signs and symptoms and according to the basic pharmacological action of the drugs[1,7] Knoefel *et al*[7] have classified reactions according to "prolonged course with primary respiratory failure" and "short course with primary cardiac failure" Betlach[6] and Adriani[1,5] prefer to distinguish between "neurological and "circulatory reactions Cullen[2] prefers to classify them according to onset "immediate" and "delayed" On the other hand Sadove *et al*[3] and Collins[4] classify them according to central nervous system effects, peripheral effects allergic responses and miscellaneous reactions A satisfactory working classification for the material contained in this chapter may be found in Table I, page 7

Systemic Reactions to Local Anesthetic Agents vs Systemic Reactions to Vasoconstrictor Drugs—Solutions containing local anesthetic agents with or without vasoconstrictor drugs are potentially toxic systemically whether administered topically, orally or by injection When a local anesthetic agent and a vasoconstrictor are mixed together it *is not* always immediately apparent which drug has caused the systemic reaction If the reaction is characterized by convulsions respiratory or cardiovascular collapse singly or together there can be little doubt that the anesthetic agent must be responsible On the other hand mild reactions of a transient nature such as excitement talkativeness or incoherent speech—the usual signs of cortical stimulation—reflect a systemic reaction either to the anesthetic drug to the vasoconstrictor drug or to both of these It is the author s opinion as well as *Bonica s*[8] that the vasoconstrictor agent not the local anesthetic agent is responsible for the signs of cortical stimulation in most of these mild reactions (see Chapter 3 page 38) *Nevertheless* a physician must never discredit the patient s history of a previous reaction to local anesthetic solutions without ascertaining the circumstances involved

Such mild and mostly transient reactions

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mouth, possible dilatation of the pupils, and even rarely atropin convulsions, is hyperergic to atropin. He *reacts in the normal way but overreacts*. A person who after taking atropin reacts with urticaria or vasomotor rhinitis re*acts in an altered manner* and is allergic.

"The person who sunburns extremely easily is hyperergic to the actinic rays. The one who following similar exposure reacts with urticaria is allergic. The person who reacts in a normal manner but more easily or more intensely is hyperergic. The term implies increased reactivity. To me this is the logical significance of the term hypersensitiveness and it would be in this sense that I would prefer to use the latter, but since it has been used so widely as a synonym for allergy or anaphylaxis, it would seem more rational to avoid its use altogether, substituting another equally appropriate term."

Other terms which should be clearly defined are *relative toxicity, relative potency* and *anesthetic index*. The relative toxicity of a local anesthetic drug used for infiltration refers to the minimum lethal dose (M.L.D.) of that drug in relationship to the minimum lethal dose of Novocain (procaine). The minimum lethal dose of Novocain is that weight of Novocain per kilogram of mouse which, when injected subcutaneously into healthy white mice weighing 16 to 24 grams which have not been fed for 6 to 8 hours, will kill two out of three mice.[17] The experiment is carried out in a temperature of 65 to 80 degrees Fahrenheit. From tests conducted on over 300 mice the minimum lethal dose of Novocain has been determined as 800 ± 10 mg. per kg.[17] Therefore, to determine the relative toxicity of another local anesthetic agent, its minimum lethal dose is compared to that of Novocain.

$$\frac{\text{M.L.D. Novocain}}{\text{M.L.D. New Drug}} = \text{RELATIVE TOXICITY}$$

For instance, a compound the minimum lethal dose of which is double that of Novocain would have a relative toxicity of 0.5, i.e., it would be half as toxic as Novocain.

By experiments, the minimum potent dose (M.P.D.) of a local anesthetic drug may be determined and compared with the minimum potent dose of Novocain. The equation for calculation of the potency of the compound relative to Novocain is

$$\frac{\text{M.P.D. Novocain}}{\text{M.P.D. New Drug}} = \text{RELATIVE POTENCY}$$

When the relative potency is high and the relative toxicity is low, it follows that the margin of safety will be high. The comparison of the relative potency to the relative toxicity is called the anesthetic index of the drug and thus, theoretically at least, *is the real measure of the safety of an anesthetic drug*. It is calculated by the following equation

$$\frac{\text{Relative Potency}}{\text{Relative Toxicity}} = \text{ANESTHETIC INDEX}$$

ETIOLOGY

The material contained in this chapter from this point on is concerned only with systemic reactions to a high blood level of the local anesthetic drug, which is by far the most common type of systemic toxic reaction to these drugs. A discussion of systemic allergic reactions to local anesthetic drugs is found in Chapter 2, page 33, while systemic reactions to vasoconstrictor drugs are reviewed in Chapter 3, page 37.

Whether or not a patient exhibits a high blood level type of reaction to a normal dose of a local anesthetic drug depends on the individual tolerance of the patient to the drug injected.[2, 3, 16] The blood level which an agent attains is directly related to the rates of absorption, detoxification and elimination of the drug. These are influenced by the following factors.

Relative Toxicities of Local Anesthetic Drugs.—Since Novocain (procaine) is chosen as a standard for infiltration and cocaine for topical application, these drugs are assigned the number 1 (see Table III, page 10, and Table IV, page 10). Cocaine is used as the standard when considering topical anesthesia because Novocain is a very poor topical anesthetic unless used in large amounts and high concentrations.[5]

It can be seen from Tables III and IV that milligram for milligram one drug may have a higher relative toxicity than another, but it

TABLE II

CLARIFICATION OF TERMS

I Allergy is a condition of unusual or exaggerated specific susceptibility to a substance which is harmless in similar amounts for the majority of members of the same species This term embraces all types of human hypersensitiveness [15]

A *Anaphylaxis* is an unusual or exaggerated reaction of the organism to foreign protein or other substances As now used the term is restricted to a *condition of sensitization in laboratory animals* produced by the injection of foreign matter such as horse serum [15]

B *Idiosyncrasy* is individual and peculiar susceptibility to some drug protein or other agent [15]

C *Susceptibility* is the state of being readily affected or acted upon The condition may be acquired familial individual inherited racial specific etc, the same as immunity [15]

D *Hypersensitivity* is having the specific or general ability to react with characteristic symptoms to *application or contact with certain substances* (allergens) in amounts innocuous to normal individuals [15]

II High Blood Level

A *True overdosage* is a blood level of a local anesthetic drug which is greater than that which the majority of patients will tolerate

B *Relative overdosage* is a blood level of a local anesthetic drug which is not excessive for the average patient but is excessive for a few patients i e hyperergy (see page 8)

agents However these terms should not be used loosely and since the general term *allergy* includes all types of sensitivity it has been selected as the term of choice for this book

A true allergy to a local anesthetic agent is characterized by one or more of the following dermatitis angioneurotic edema hypotension, wheals itching, asthmatic breathing and "clinical anaphylactic shock" Only reactions which may occur following the use of small or infinitesimal amounts of local anesthetic drugs and which are characterized by these signs and symptoms can automatically be termed allergic This does not mean that allergy cannot result from a large dosage of a local anesthetic drug—clearly if small doses cause an allergic reaction in a particular patient large doses would have the same result Nevertheless, when a patient reacts to a large dosage (25 to 50 cc of 2% Novocain) the reaction cannot be called "allergic" unless he reacts similarly when tested with small doses (1 to 3 cc) of the same drug administered in the same concentration and fashion *A systemic toxic reaction to a high blood level of a local anesthetic drug is not an allergy* although conceivably both types of reaction could occur simultaneously

The term *high blood level* in this book indicates an overdosage of the drug for a particular individual This may be either true or relative A *true* high blood level is caused by one or all of the following (1) too large a dosage of the local anesthetic drug (2) unusually rapid absorption of the drug (3) unusually slow detoxification or (4) slowed elimination A *relative* high blood level is one in which the level may be high for a particular individual or a few individuals but not high for the greater majority of patients These few patients may be blocked successfully with smaller amounts of the local anesthetic agent than usual without a reaction It should also be noted that identical amounts of a local anesthetic solution administered in the same way to one individual at different times may at one time cause no reaction and at another time cause a reaction depending on the patient's physical status For example when a person is rested and in excellent physical condition he may tolerate the average dose of a drug with no difficulty However should this patient be worried up all night and exhausted he might not tolerate this dosage without an untoward reaction Vaughan and Black[16] term a relative high blood level "hyperergy" and state "This term has been proposed by the writer as indicating an increased sensitiveness or reaction capacity to a foreign substance for example drugs There are persons who react much more strongly to the usual dose of atropin or belladonna than do the majority The person who with the normal dose develops an excessively dry

From Table V, page 11, it appears that in concentrations of 0.5% or less, a larger dose of Xylocaine than Novocain can be used but in concentrations above 0.5% the lethal doses of Xylocaine are equal to or smaller than those of Novocain.

The figures for the toxicity, potency and anesthetic index for Cyclaine (hexylcaine) another recently introduced local anesthetic agent, are not available at the present.

Total Weight of the Drug Injected.—Through clinical experiments there has been determined the approximate maximum dose of a local anesthetic drug which may usually be injected into or applied to the mucous membranes of the healthy adult without causing an untoward reaction (see Table VI, page 12). Amounts of the drug in excess of these figures are to be considered an overdosage because an untoward reaction will very probably occur. In most instances regional procedures may be executed effectively with amounts less than the maximum dosage provided the technique used is correct in all details. In the debilitated patients, the undernourished, those at the extremes of age, and those whose physical status is affected by disease, the total quantity of the drug injected must be reduced substantially from the maximum dosage if reactions are to be avoided. In these substandard risks the detoxification mechanisms of the body are often impaired. The liver is believed to be the site of detoxification of most local anesthetic agents, but some drugs, like cocaine, are excreted by the kidney unchanged.[5, 20]

Concentration of the Solution.—The toxicity of a local anesthetic drug increases in geometrical not arithmetical progression with increase in concentration.[21, 22] For example, 120 cc. of a 1% solution of Novocain will kill a rat in 20 minutes, while only 10 cc. of a 2% solution will produce the same lethal effect; that is, with a weak solution a dose of 1.2 grams of Novocain is lethal, while only 0.8 gram of Novocain kills when employed in concentrated form.[4]

This indicates the importance of using the weakest concentration which will give the required analgesia. In general, large nerve trunks require higher concentrations of the agent than do skin infiltrations. Gasser and Erlanger[24] point out that sensory fibers are paralyzed by one tenth the strength of local anesthetic solutions needed to block motor fibers. However, irrespective of the concentration employed, the recommended maximum dose of the agent should not be exceeded (see Table VI, page 12).

Vascularity of Area Injected.—The vascularity of a region into which a solution is injected governs to a large extent the rapidity with which the agent is absorbed into the blood stream.[25, 28] This may explain why the topical use of Pontocaine on the mucous membranes of the mouth, nose, pharynx, larynx

TABLE V

TOXICITY OF XYLOCAINE COMPARED WITH NOVOCAIN, PONTOCAINE, COCAINE
(From Gordh[19])

Animal	*Concentration*	*Lethal dose (LD 50) g./kg.**			
		Xylocaine (Lidocaine)	*Novocain (Procaine)*	*Pontocaine (Tetracaine)*	*Cocaine*
Mouse (subcut.)	0.5%	1.07	1.02	0.05–0.10	0.10–0.20
	1%	0.72	1.00		
	2%	0.59	0.90		
	4%	0.42	0.83		
	8%	0.33	0.63		
Rabbit (subcut.)	2%	0.08–0.10	0.10–0.20		

*The lethal dose varies with the concentration and for low concentrations may vary at a higher rate.

also may produce effective anesthesia in greater dilutions, i e it has a greater relative potency therefore its anesthetic index would be greater, and in clinical use it might actually prove to be less toxic While the anesthetic index of a drug as shown in Tables III and IV will help to evaluate it we must realize that figures determined in this manner may give only an approximate idea as to what might be expected of the use of that drug in man As can be seen in Table III the toxicity of a drug was obtained by animal experiment while the potency was determined in humans While the anesthetic index of Pontocaine determined in this fashion would indicate that the drug is slightly more toxic than Novocain when used for local infiltration and peripheral nerve block it has been the clinical experience both of the author and Bonica that in man this is not the case [79, 34] Species variations are difficult to assess and if a correct evaluation of a drug s anesthetic properties is to be made its action in man is the ultimate criterion

Even if the same species is used the method of administering the drug must be the same and the experiment conducted under identical circumstances i e temperature etc When toxicity and potency are not studied by the same method their comparison may be misleading e g if a drug is to be evaluated for topical analgesia both its toxicity and potency should be studied with topical applications To investigate potency by topical application and toxicity by subcutaneous injection may lead to unreliable statistics Moreover even if the anesthetic index of a drug determined in any fashion were reliable other properties of the drug e g duration of action might indicate its use in a particular case

The specific figures for toxicity potency and anesthetic index for Xylocaine (lidocaine) a basic amid and a relatively new local anesthetic drug which is being used widely both topically and parenterally were not available in the American literature and are not included in Tables III and IV However, Lofgren [18] states that (1) milligram for milligram the toxicity of Xylocaine is greater than that of Novocain (2) its efficiency is greater than that of Novocain, and (3) the anesthetic index is greater than that of Novocain

TABLE III

RELATIVE TOXICITIES AND POTENCIES OF LOCAL ANESTHETIC DRUGS COMMONLY USED FOR INFILTRATION AND BLOCK ANALGESIA
(From Collins[4])

Drug	*Toxicity Relative to Procaine (Subcutaneous Mouse)*	*Potency Relative to Procaine (Intradermal Man)*	*Anesthetic Index*
Novocain (procaine)	1	1	1 0
Intracaine (diethoxin)	1	4 3	4 0
Metycaine (piperocaine)	3	2 3	0 8
Cocaine	4	4	1 0
Pontocaine (tetracaine)	12	5–10	0 8
Nupercaine (dibucaine)	20	22–25	1 2

TABLE IV

RELATIVE TOXICITIES AND POTENCIES OF LOCAL ANESTHETIC DRUGS COMMONLY USED FOR TOPICAL ANESTHESIA
(From Collins[4])

Drug	*Toxicity Relative to Cocaine (Subcutaneous Mouse)*	*Potency Relative to Cocaine (Corneal Anesthesia)*	*Anesthetic Index (Potency—Toxicity)*
Cocaine	1	1	1 0
Intracaine (diethoxin)	0 5	1	2 0
Pontocaine (tetracaine)	3–5	10 0	2 5
Nupercaine (dibucaine)	5	100 0	20 0

artery during a stellate ganglion or cervical block may result in convulsive seizures, etc., while the same amount of the drug deposited intravascularly during a caudal injection may produce only dizziness.

Direct Action of the Local Anesthetic Agent on the Blood Vessels.—Most anesthetic agents, i.e., Novocain, Pontocaine, Xylocaine, Metycaine, Intracaine, and Nupercaine, are vasodilators and this property increases the rate of their absorption.[33] On the contrary, cocaine is a strong vasoconstrictor and therefore absorbs slowly.

Use of Vasoconstrictor Agent.—These agents by causing local constriction of the blood vessels—the so called "chemical tourniquet effect"—decrease the rate of absorption of drugs used in local anesthetic solutions, thus lowering the incidence of reactions.[29, 34, 36] The decrease in absorption also prolongs and potentiates the anesthesia in most instances.

Rate of Injection of the Solution.—A rapid rate of injection of large volumes and/or high concentrations of local anesthetic solutions augments absorption, resulting in higher blood levels. Often relatively large quantities

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TABLE VI—*Continued*

DRUGS COMMONLY USED FOR REGIONAL ANALGESIA

Drugs	*Uses and Dosage (Maximum)*			
	Topical	*Infiltration*	*Spinal*	*Epidural (Caudal or Lumbar Area)*
Intracaine *B* diethoxin *USP*	Nose, pharynx, trachea: not commonly used Urethra: not commonly used	200 cc of 0.5% = 1000 mg 100 cc of 1.0% = 1000 mg 50 cc of 2.0% = 1000 mg	3 cc of 5% = 150 mg. Not commonly used	50 cc of 1% = 500 mg
Nupercaine *B* dibucaine *NF* Percaine *B* cinchocaine *BP*	Nose, pharynx, trachea: not commonly used Urethra: 15 cc of 0.2% = 30 mg (High toxicity)	200 cc of 0.02% = 40 mg 80 cc of 0.05% = 40 mg 40 cc of 0.10% = 40 mg Not commonly used	20 cc of 0.5% or 4 cc of 0.25% = 10 mg	Not used
Cocaine *USP*	Nose, pharynx, trachea: 4 cc of 5% = 200 mg Urethra: 30 cc of 1% = 300 mg (High toxicity)	Not commonly used	Not commonly used	Not used
Cyclaine *B* hexylcaine *NNR*	Nose, pharynx, trachea: 10 cc of 5% = 500 mg Urethra: 10 cc of 5% = 500 mg or 15 cc of a 5% jelly = 750 mg	100 cc of 0.5% = 500 mg 50 cc of 1.0% = 500 mg 25 cc of 2.0% = 500 mg	4 cc of 1.25% = 50 mg N.B. Usually the solution contains 5% dextrose	50 cc of 1% = 500 mg

BP = British Pharmacopeia. *USP* = United States Pharmacopeia. *NF* = National Formulary (U.S.A.). *NNR* = New and Non-official Remedies (U.S.A.). *B* = Brand Names. *BS* = British Synonym. *BPC* = British Pharmacopeial Commission Approved Name. Code adopted from Geddes, I. C.: A Review of Local Anesthetics. *Brit. J. Anes.*, 25:208-244, 1954.

and trachea—all areas of great vascularity—may result in a high percentage of systemic reactions while an injection of the same drug in local infiltration or nerve block procedures results in very few untoward reactions.[79, 37]

During nerve block and local infiltrations the areas of high vascularity which must be injected with care are the scalp, face, neck, corpus cavernosa, and epidural space (particularly the sacral area). Also because the blood supply to inflamed areas is greatly increased, it is best to avoid injecting them with local anesthetic agents not only because the possibility of a toxic reaction is enhanced but also because (1) the procedure might spread the infection, (2) it is painful, and (3) the resulting anesthesia is often poor.

The topical application of a local anesthetic drug to an inflamed mucous membrane, particularly the urethra, is singularly dangerous since systemic toxic reactions of a high blood level type will very probably ensue.

Inadvertent Intravenous Injections.—When drugs are inadvertently injected into the blood stream, a high blood level rapidly results. *Arterial injections may be more dangerous than venous injections, but either is serious* (see Figure 1, page 14). The region in which the intravascular injection is made may also determine to some extent the severity of a reaction. For example, 5 to 7 cc of a local anesthetic solution injected into the vertebral

TABLE VI

DRUGS COMMONLY USED FOR REGIONAL ANALGESIA

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	Topical	*Infiltration*	*Spinal*	*Epidural (Caudal or Lumbar Area)*
Novocain *B* procaine *USP BP* ethocaine *BS* Kerocaine *B* Planocaine *B* Scurocaine *B* Neocaine *B*	Ineffective	200 cc of 0.5% = 1000 mg 100 cc of 1.0% = 1000 mg 50 cc of 2.0% = 1000 mg	4 cc of 5% = 200 mg	50 cc of 2% = 1000 mg
Pontocaine *B* tetracaine *USP BS* amethocaine *BP* Anethaine *B* Decicain *B* Pantocaine *B* Butethanol *B*	Nose, pharynx, trachea: 4 cc of 2.0% or 8 cc of 1.0% = 80 mg Urethra: not commonly used	1 mg per pound body weight—do not exceed 200 mg or a concentration of 0.25%	2 cc of 1% = 20 mg	50 cc of 0.15% = 75 mg
Xylocaine *B* lidocaine *NNR* lignocaine *BPC* Xylotox *B*	Nose, pharynx, trachea: 10 cc of 2% = 200 mg Urethra: 15 cc of 2% jelly = 300 mg	100 cc of 0.5% = 500 mg 50 cc of 1.0% = 500 mg 25 cc of 2.0% = 500 mg	Not established; probably 4 cc of 4% = 160 mg	50 cc of 1.0% = 500 mg 25 cc of 2% = 500 mg
Metycaine *B* *piperocaine USP BPC*	Nose, pharynx, trachea: not commonly used Urethra: 30 cc of 2% = 600 mg	200 cc of 0.5% = 1000 mg 100 cc of 1.0% = 1000 mg 65 cc of 1.5% = 1000 mg	4 cc of 4% = 160 mg	50 cc of 1.5% = 750 mg

centrations and volume of an anesthetic solution are employed the addition of hyaluronidase (Diffusin, Wydase, Alidase, etc), a spreading agent, increases the chances of a toxic reaction to the anesthetic, to the vasoconstrictor or to both[37] Even though a vasoconstrictor drug is incorporated in the solution to retard absorption the spreading factor allows an increase of up to 40% above the normal anesthetized area (see Figure 2, page 15) Since greater surface permits more rapid absorption the blood level of the local anesthetic agent and vasopressor drug would be higher than normal in a shorter period of time when hyaluronidase has been added

Patient's Physical Status—To a marked extent the physical status of a patient may determine his susceptibility to reactions from local anesthetic solutions As mentioned above, the liver is thought to detoxify many local anesthetic agents, therefore, perhaps the concentration and volume of these drugs should be reduced for patients with liver disease Other physical factors which may increase the incidence of reactions are hyperpyrexia, debility, shock, starvation lowered metabolism and the extremes of age

Some diseases, on the other hand, may make the patient more resistant Thyrotoxicosis with its associated high metabolic rate may result in a more rapid detoxification of the drug while arteriosclerosis may retard absorption of injected solutions

The effect of vitamin C deficiency in increasing a patient's susceptibility to a local anesthetic drug has been pointed out by Richards[39] He has shown that starvation and vitamin C deficiency increase the instances of



Figure 2 Effects of hyaluronidase on the diffusion of 2 cc of a Diodrast local anesthetic solution placed subcutaneously *Left* Solution without hyaluronidase *Right* Solution containing hyaluronidase

of local anesthetic solutions may be administered to a patient, provided this is done over a long period of time or the intervals between injections are long enough to allow for detoxification For example 2 to 3 grams of Novocain may be given intravenously by a slow drip over a 12 or 24 hour period without causing a systemic reaction, but if more than 1 gram is used while performing a local infiltration or nerve block procedure, a systemic toxic reaction may easily result

Spreading Agents.—Provided the usual con



Figure 1 Arteriogram from a test dose of 2 cc of a Diodrast local anesthetic solution inadvertently deposited in the vertebral artery This complication occurred during the execution of a stellate ganglion block although attempts to aspirate blood were unsuccessful. It is our custom in this block procedure to inject a 2 cc test dose and wait 15 to 30 seconds before discharging another 8 to 10 cc of the solution into the area of the stellate ganglion The 2 cc deposited in the artery made the patient complain immediately of dizziness of the feeling of "blacking out" and of nausea Had the total dosage been injected, a severe systemic toxic reaction would probably have ensued.

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alone by bag and mask. In this one case the convulsion was attributed to oxygen want. Ten of these 14 cases were heavily medicated and it is possible that the premedication prevented or altered the early signs of cerebral stimulation but we doubt this. A number of German investigators after extensive studies on Pontocaine in a variety of laboratory animals and man also found that respiration usually failed before the circulation did.[4, 7]

In addition these investigators found that toxic doses in animals caused disturbances in cardiac rhythm which always disappeared without myocardial damage. Clinically we have also observed that cardiac irregularities may occur, for in 10 of our patients receiving Pontocaine (50 cc. of 0.25%) an irregular pulse appeared as the only sign of an untoward reaction to the drug. So far this has corrected itself within 10 minutes without treatment or with the administration of oxygen only. In only five cases in over 8,000 local infiltrations and nerve blocks using Pontocaine have we seen diffuse sweating, nausea, vomiting, palpitation, pallor, and tachycardia which so often follow the injection of Novocain. The fact that such changes are infrequent when Pontocaine is employed in local infiltration and nerve block procedures is further substantiated by a number of authors whose combined cases total over 31,000.[26, 28, 30, 54, 55]

Certain writers have emphasized that systemic toxic reactions to Cyclaine seldom occur.[56, 57] However, at the Mason Clinic the injection of 30 cc. of a 2% concentration of the drug into the caudal canal of 100 obstetrical patients was followed by such reactions in three cases, each of which progressed to convulsions. These reactions were evidently due to absorption of the drug, not an inadvertent intravascular injection of the entire 30 cc., because a level of anesthesia was established which ranged from the pubic bone to the xiphoid process.

Onset of Signs and Symptoms.—While the signs and symptoms of a systemic reaction usually fit a typical pattern, i.e., progress slowly from the signs of cortical stimulation (restlessness, etc.) to the signs of severe depression and death, depending on the rapidity of recognition and therapy, the following variations may occur: (1) all the signs and symptoms may occur simultaneously, (2) one sign may predominate, the others being less obvious, and (3) the reaction may occur insidiously so that, if the patient is not being carefully observed, death may occur without warning. In this book, for clarity, the signs and symptoms reflected in each system, i.e., nervous, circulatory, etc., will be considered separately, with full realization that they may occur either individually, progress slowly from one system to the other, or all occur at one time.

The onset of a systemic reaction to a high blood level of the local anesthetic drug may be immediate or delayed. The reaction may occur seconds to minutes after the start of the injection with all the signs and symptoms occurring at once. Such rapid onset is due to an inadvertent intravascular injection of the anesthetic solution or to rapid absorption of a large quantity of the agent in a very short period of time. There is no slow, progressive deterioration of the patient's condition, collapse is rapid and total. Death often results before treatment can be instituted. This type of reaction, where *large quantities* of the drug are used, should not be confused with the allergic "clinical anaphylactic shock" type occurring from relatively *infinitesimal amounts* of a drug.

On the other hand the reaction may occur 5 to 30 minutes after the injection. Fortunately, this is the more frequent of the two types of systemic reactions from a high blood level of the anesthetic drug. It is caused by a slow build-up of a toxic blood level and it usually progresses in stages as listed below, the cortical signs first, then the signs of cardiovascular collapse.

Nervous System (Cortical) Signs and Symptoms.—The early signs and symptoms of cortical stimulation are one or a combination of the following: restlessness, nervousness, apprehension, pugnaciousness, unreasonableness, loquacity, incoherent speech, headache, dizziness, blurred vision, metallic taste, roaring in

systemic reaction in animals He believes that patients with poor physical status should receive an infusion of dextrose with ascorbic acid before an injection of a local anesthetic

Environmental Temperature — The environmental temperature according to Collins,[4] plays an important role in increasing the incidence of reaction He states that high temperatures increase absorption and diffusion of injected drugs This may be a significant point to remember when administering local anesthetic drugs during the summer in the tropics and to patients with a high fever However the experience of the author both in the army and in civilian practice would indicate that environmental temperature is not of great enough significance to warrant changing the dosages in Table VI, even in exceptionally warm climates

SIGNS AND SYMPTOMS

Basically, the signs and symptoms of a systemic toxic reaction due to the high blood level of the local anesthetic drug whether administered parenterally, orally or topically are referable to the central nervous system and perhaps the cardiovascular system They are first those of stimulation i e restlessness tremors and convulsions and secondly those of depression following overstimulation Death is usually attributed to respiratory depression[3 0] However according to Beck and Mautz[39] Burstein[40] Burstein and Rovenstine[41] Graubard and Peterson[42] and Steinhaus[43] local anesthetic drugs may cause depression of the heart when applied directly Therefore while death is usually attributed to respiratory depression the direct action of these drugs upon the heart cannot be completely ignored as contributing to cardiac depression and collapse

Goodman and Gilman[20] state " it is highly significant that all the local anesthetics produce the same type of symptoms when given in toxic amounts However it is interesting to note that while this may be true experimentally it may not always hold true clinically Toxic reactions in human beings from Novocain Intracaine, cocaine Nupercaine Xylocaine and Cyclaine in most instances follow the common pattern of a reaction, i e the onset is slow with the minor signs of cerebral stimulation slowly progressing to convulsions and peripheral vascular collapse Nevertheless, sometimes reactions to Xylocaine may not be signalled by cerebral stimulation When doses of 0 5 gm or more of Xylocaine are employed central depression without any other signs or symptoms of a toxic reaction may be noted These patients will not respond to questioning and appear to be in the first plane of surgical anesthesia Their course during this is usually uneventful blood pressure, pulse, and respirations remain unchanged and within one half to three fourths of an hour they regain consciousness This type of reaction may also follow the use of large amounts of Novocain[44]

While reactions to Pontocaine may follow the above mentioned characteristic pattern we have found that most of the reactions observed from this drug were atypical At the Mason Clinic in over 6 000 cases in which Pontocaine has been used for regional block other than subarachnoid (spinal) and epidural (peridural) block 14 major reactions attributed to this drug have been observed The reactions in these cases were not heralded by restlessness nervousness dizziness sweating pallor and tachycardia As a matter of fact, there was no such warning They occurred 5 to 10 minutes after the completion of the block and were characterized by drowsiness shallow respiration loss of consciousness and apnea Blood pressure and pulse did not vary in these patients until apnea occurred and then they began to increase When artificial respiration with oxygen by bag and mask was instituted the blood pressure and pulse returned to normal and remained so Operations were not cancelled in any of these cases but were delayed for 30 to 45 minutes until the patients regained consciousness and started to breathe voluntarily Only one of these patients who was not being carefully observed started to convulse and this was corrected by the administration of oxygen

no initial variation in the blood pressure and pulse from that determined prior to the injection However, as respirations become shallow or cease the blood pressure rises and a tachycardia ensues This resembles a reaction to a vasoconstrictor drug and may lead to confusion (see Chapter 3 page 38) Such a reaction reflects either oxygen want or initial stimulation of the cardiovascular center in the medulla by the local anesthetic agent As the reaction progresses, the hypoxia becomes more severe, either from apnea or from the direct depressant effect of the local anesthetic agent on the cardiovascular center or the heart and blood vessels, the blood pressure begins to fall, and a bradycardia appears If the hypotension is not corrected the patient usually becomes more cyanotic he perspires profusely, the skin is clammy and there ensues syncope and peripheral vascular collapse with a weak and often unobtainable pulse At least from a pharmacologic standpoint the hypotension which results from this type of reaction has usually been blamed on depression of the vital centers in the medulla as a result of previous overstimulation [3, 20] However, in light of recently published articles the question arises as to whether this is the only responsible factor [1, 4, 33, 39, 43] These latter reports make it seem possible that hypotension which develops with the oxygen want might be at least partially caused by a direct action of the local anesthetic agent on the myocardium and/or the peripheral vessels The authors of a number of these articles stress that with the exception of cocaine most local anesthetic agents given in adequate doses intravenously or applied in sufficient quantities directly on the heart may depress heart action and bring about bradycardia decreased cardiac output and eventually cardiac standstill [39, 42] Others have pointed out that local anesthetic drugs, cocaine being the exception are potent vasodilators and act directly on the peripheral vessels [1, 4, 33] Nevertheless until further experimentation proves otherwise it must be assumed that the oxygen want during a local systemic reaction is primary and that the cardiovascular collapse is secondary to it i e oxygen want is the cause of hypotension If the oxygen want hypotension and syncope are not corrected within two to ten minutes cardiac failure may result

Cardiac Failure and/or Death—When cardiac failure and/or death occur from a toxic dose of a local anesthetic drug it is presumably caused by depression of the respiratory center from previous overstimulation by the drug with cardiac failure as a secondary phenomenon [3, 20] This theory is based on the fact that the brain being the most sensitive organ of the body, would be the first to respond to *small* amounts of the local anesthetic agent in the blood

However when a *large* dose of the agent is injected inadvertently into the blood stream or rapid absorption from mucous membranes occurs, the possibility of cardiac failure with respiratory arrest as a *secondary* phenomenon cannot be completely overlooked Under these circumstances, with the exception of an intra-arterial injection of the neck and head the first organ to be reached by a massive dose of the drug would be the heart This could explain the fatalities which occur during or immediately following an administration particularly when convulsions etc are absent Further research would be helpful to ascertain what part direct cardiac depression from the local anesthetic plays in the picture of systemic toxic reactions but until this is done oxygen want must be considered the cause of circulatory depression

Irrespective of the cause cardiac failure does occasionally occur when regional analgesia is used and must be diagnosed promptly and treated immediately if death is to be avoided The physician must be able to recognize its signs and symptoms (see Chapter 7 page 73)

PROPHYLAXIS

While it is difficult to prevent allergic systemic reactions from infinitesimal amounts of local anesthetic agents those caused by a high blood level of these drugs from overdosage may usually be reduced to a minimum by observing the following prophylactic measures

the ears nausea and vomiting choreiform movements, tremors and twitchings The metallic taste, dizziness blurred vision and roaring in the ears have been the most prominent of these early cortical signs and symptoms in the author s experience If the reaction does not progress past this initial stage these signs and symptoms are of little clinical importance However, they do announce the fact that a toxic reaction exists and thereby warn that the reaction may progress to unconsciousness convulsions nausea and vomiting cardiovascular collapse and respiratory collapse Convulsions indicate marked stimulation of the cortex with associated increased metabolism increased oxygen demand and since respirations usually cease during a convulsion hypoxia or anoxia results

Nausea and/or vomiting may occur either early or late in the course of a systemic toxic reaction to a local anesthetic drug and may not indicate the severity of the reaction The vomiting center may be stimulated by hypoxia by the excessive blood level of the local anesthetic agent, or by a combination of both The seriousness of this complication will, in most instances depend on the preoperative preparation and cooperation of the patient.

In the patient who has been prepared for an elective block procedure by restriction of the oral intake and/or by Levin tube suction of the stomach stimulation of the vomiting center usually presents less of a problem as the stomach contains little or nothing to expel When intake has been restricted the volume regurgitated in most instances is small and the patient will be able to expel it before he takes an inspiration However the patient whose oral intake has not been restricted and whose stomach has not been pumped presents a greater problem The volume of stomach contents is usually large and more likely than not will contain particles of undigested food If he vomits he may not be able to expel a large volume of vomitus from the oral cavity before he takes a breath In that case he aspirates vomitus and the problem is twofold the hydrochloric acid damages the lungs and the food particles barricade the passage of air or oxygen into the trachea bronchi and/or alveoli If the patient is conscious and will cooperate, there may be no difficulty in clearing the airway, but if he is uncooperative or unconscious much difficulty may be encountered (see Chapter 18 page 165)

Respiratory System Signs and Symptoms — In the very early stages of a reaction the occasional patient may show an increase in the rate and/or depth of respiration In almost all cases irregular respiratory rate and excursion sighing dyspnea periods of apnea and finally complete respiratory arrest are encountered as a manifestation of depression of the respiratory center in the medulla presumably from its previous overstimulation[3, 20] Nevertheless the part played by tissue hypoxia from the hypotension which invariably accompanies the late stages of this type of reaction cannot be ignored completely Inasmuch as depressed respirations or apnea usually precede circulatory collapse the patient becomes cyanotic i e, there is 5 gm or more of unreduced hemoglobin present per 100 cc of blood and since the circulation is reaching the periphery particularly the dermis, cyanosis is present While the convulsions which attend this type of reaction are usually attributed to overstimulation of the cortex by the local anesthetic drug they may be the result of hypoxia or at least the hypoxia may have an additive effect

On rare occasions the lungs may show signs of hypersecretion râles rhonchi, bronchospasms and/or an asthmatic type of respiration Of course, a large dosage of the local anesthetic drug would lead us to suspect that these must be signs of a high blood level systemic reaction but the use of a large dosage does not *preclude* the possibility that an allergic reaction has taken place (see page 8) It is not clear at present whether these signs should be classified as an allergic systemic reaction or as a high blood level systemic reaction

Cardiovascular System Signs and Symptoms — *When a reaction starts there may be*

no initial variation in the blood pressure and pulse from that determined prior to the injection. However, as respirations become shallow or cease the blood pressure rises and a tachycardia ensues. This resembles a reaction to a vasoconstrictor drug and may lead to confusion (see Chapter 3 page 38). Such a reaction reflects either oxygen want or initial stimulation of the cardiovascular center in the medulla by the local anesthetic agent. As the reaction progresses the hypoxia becomes more severe either from apnea or from the direct depressant effect of the local anesthetic agent on the cardiovascular center or the heart and blood vessels the blood pressure begins to fall and a bradycardia appears. If the hypotension is not corrected the patient usually becomes more cyanotic he perspires profusely the skin is clammy and there ensues syncope and peripheral vascular collapse with a weak and often unobtainable pulse. At least from a pharmacologic standpoint the hypotension which results from this type of reaction has usually been blamed on depression of the vital centers in the medulla as a result of previous overstimulation[2,20]. However in light of recently published articles the question arises as to whether this is the only responsible factor[1,4,33,39,43]. These latter reports make it seem possible that hypotension which develops with the oxygen want might be at least partially caused by a direct action of the local anesthetic agent on the myocardium and/or the peripheral vessels. The authors of a number of these articles stress that with the exception of cocaine most local anesthetic agents given in adequate doses intravenously or applied in sufficient quantities directly on the heart may depress heart action and bring about bradycardia decreased cardiac output and eventually cardiac standstill[39,42]. Others have pointed out that local anesthetic drugs cocaine being the exception are potent vasodilators and act directly on the peripheral vessels[1,4,33]. Nevertheless until further experimentation proves otherwise it must be assumed that the oxygen want during a local systemic reaction is primary and that the cardiovascular collapse is secondary to it i.e. oxygen want is the cause of hypotension. If the oxygen want hypotension and syncope are not corrected within two to ten minutes cardiac failure may result.

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However when a *large* dose of the agent is injected inadvertently into the blood stream or rapid absorption from mucous membranes occurs the possibility of cardiac failure with respiratory arrest as a *secondary* phenomenon cannot be completely overlooked. Under these circumstances with the exception of an intraarterial injection of the neck and head the first organ to be reached by a massive dose of the drug would be the heart. This could explain the fatalities which occur during or immediately following an administration particularly when convulsions etc. are absent. Further research would be helpful to ascertain what part direct cardiac depression from the local anesthetic plays in the picture of systemic toxic reactions but until this is done oxygen want must be considered the cause of circulatory depression.

Irrespective of the cause cardiac failure does occasionally occur when regional analgesia is used and must be diagnosed promptly and treated immediately if death is to be avoided. The physician must be able to recognize its signs and symptoms (see Chapter 7 page 73).

PROPHYLAXIS

While it is difficult to prevent allergic systemic reactions from infinitesimal amounts of local anesthetic agents those caused by a high blood level of these drugs from overdosage may usually be reduced to a minimum by observing the following prophylactic measures

Avoid Overdosage of the Local Anesthetic Agent.—This is best accomplished by (1) using local anesthetic drugs whose toxicities have been well established by clinical and experimental use (see Table VI, page 12); (2) not exceeding their recommended toxic doses; (3) using the weakest concentration and the least volume of the drug that will accomplish the desired degree of analgesia, and (4) injecting carefully so as to not deposit a large amount of the drug intravenously.

Use of Vasoconstrictor Drugs.—These drugs constrict the blood vessels, thus slowing absorption. This so called "physiologic or chemical tourniquet" effect at the site of injection prevents rapid rises in the blood levels of drugs, prolongs the duration of anesthesia, and produces a relatively "bloodless" field at the site of injection. Therefore, when near maximum dosages of local anesthetic agents are to be employed, particularly if the area to be injected is highly vascular, it is usually efficacious to incorporate a vasoconstrictor drug in the anesthetic solution.

TABLE VII

CLASSIFICATION OF COMMONLY USED LOCAL ANESTHETIC AGENTS ACCORDING TO CHEMICAL STRUCTURE

Chemical Group	*Local Anesthetic Drug*
Para amino benzoic acid esters	Novocain (procaine) Pontocaine (tetracaine) Monocaine Benzocaine Butesin Butyn
Benzoic acid esters	cocaine Metycaine (piperocaine)
Para-ethoxy benzoic acid esters	Intracaine (diethoxin)
Beta diethyl amino ethyl amide	Nupercaine (percaine dibucaine)
w diethylaminoacet 2 6-xylidide hydrochloride	Xylocaine (lidocaine)
1-cyclohexylamino-2-propyl benzoate hydrochloride	Cyclaine (hexylcaine)

Choose the Local Anesthetic Agent Carefully.—If the patient is known to have had a systemic toxic reaction to one specific local anesthetic and a general anesthetic is contraindicated, a local anesthetic drug of another chemical group *may in most instances* be used without producing a reaction. For example, Pontocaine, Monocaine and Novocain are para amino benzoic acid esters; cocaine and Metycaine are benzoic acid esters; Nupercaine is a quinoline derivative, and Xylocaine is a basic amid (see Table VII, page 20). Therefore, if a patient shows a reaction to a drug in one of the above four mentioned chemical groups, a drug in another chemical group may conceivably be injected without causing a reaction; for example, if a reaction to Novocain occurs, Xylocaine in most instances may be used without an untoward reaction.

Use a Barbiturate Prior to Block.—Tatum, Atkinson and Collins[60] have thought that barbiturates not only arrest convulsions resulting from an overdose of local anesthetic agents in animals, but noted that when they were given prophylactically, the minimum lethal dose of the local anesthetic is increased four times. The higher forms of animals, such as monkeys and man, having a more highly developed central nervous system, are thought to be more susceptible to the toxic actions of local anesthetic agents—Tatum and Collins[61] showed that barbiturates afforded monkeys relatively more protection than the lower forms of the laboratory animals. They concluded that humans, having an even greater development of the central nervous system than do monkeys, would therefore be afforded even greater protection. A great number of publications on local anesthesia still stress the use of barbiturates prior to the injection of local anesthetic solution, based on the work of Tatum *et al*.[4, 17, 60, 67, 77, 78]

However, the following are some practical reasons why barbiturates are not given for this supposedly "protective effect" prior to all nerve block analgesia at the Mason Clinic.

First they often make the patient uncooperative and accurate acknowledgment of paresthesias often necessary for the success of some block procedures is difficult to elicit Second they depress the cortex and thereby often obscure the cortical signs and symptoms of a toxic reaction This is perfectly all right if the reaction does not progress past the point of cortical stimulation but if it does the barbiturates are then actually performing a disservice by hiding the "warning signals" of a reaction Lastly they act synergistically to *deepen the depression stage which follows* overstimulation of the brain particularly the vital medullary centers"[9] Thereby they may make the treatment of a reaction more difficult

Sadove *et al*[1] also feel that the usefulness of barbiturate prophylaxis has been greatly overrated and state "We would even go further and say that barbiturates being depressants of the central nervous system (and therefore so useful in counteracting the central nervous system stimulation type of reaction) will be decidedly harmful in those cases in which central nervous system depression follows on stimulation" Steinhaus[43] writes "Our experience in cocaine intoxication produced by constant intravenous injections revealed the antagonism of the barbiturates to be somewhat less effective than previous workers [Tatum *et al*[60]]* had reported" In addition according to Knoefel *et al*[7] barbiturates do not remove the possibility of toxic reactions and are of little use in those reactions which are a result of rapid absorption They claim "As most of the cases of procaine intoxication seem to be of the type in which prophylaxis is unavailing the practical value of administering barbiturates is small"

My associates and I use barbiturates only for their sedative effect on the patient and do not attempt to use doses large enough to prevent the signs of cortical stimulation As a matter of fact when paresthesias are to be obtained prior to injecting the drug we give no preblock barbiturate[6] However after the block has been completed if the patient wishes to be less aware of his surroundings a barbiturate usually 1½ gr (90 mg) to 3 gr (150 mg) of Nembutal (pentobarbital) is given intravenously

* Interpolation mine

Swallowing of Drugs Used Topically on Pharynx, Larynx, and Trachea —Swallowing of a local anesthetic drug applied topically to the pharynx larynx or trachea is to be discouraged according to Collins[4] He states that excess quantities should be spat out because rapid absorption of the agent from the *mucosa of the esophagus and stomach may* occur[4] Steinhaus[43] does not agree with this and states "Absorption from the esophagus and stomach does not seem to be the likely cause of reactions [to local anesthetic drugs] as judged by our results which conclusion is substantiated by the comparatively low blood levels attained with oral administration of cocaine" Our own work with the oral use of Novocain (procaine) solutions and Xylocaine (lidocaine) Viscous would support the fact that the local anesthetic agents are not rapidly absorbed from the esophagus or stomach

Use of Vitamin C (ascorbic acid) —Richards[38] concluded from animal studies that starvation and vitamin C deficiencies increased the incidence of systemic toxic reactions to local anesthetic drugs in animals Consequently he postulated that the debilitated and cachectic patient should receive infusions of dextrose and ascorbic acid prior to injection of a local drug To date the author has never done this and has noted no greater incidence of reactions in this group of patients than in the young healthy vigorous adult probably because the dosage of the drug was individualized

Skin Wheals, Lozenge, Patch and Mucous Membrane Tests —The efficacy of tests to ascertain beforehand whether or not a patient will exhibit a high blood level type of a systemic toxic reaction to that drug is debatable Ryan and Shouldice[63] state "For the first 7 000 cases [regional blocks with procaine] an intradermal test with 2% procaine hydrochloride was carried out preoperatively Usually there was no skin reaction, but in a number of

Avoid Overdosage of the Local Anesthetic Agent—This is best accomplished by (1) using local anesthetic drugs whose toxicities have been well established by clinical and experimental use (see Table VI page 12) (2) not exceeding their recommended toxic doses (3) using the weakest concentration and the least volume of the drug that will accomplish the desired degree of analgesia and (4) injecting carefully so as to not deposit a large amount of the drug intravenously

Use of Vasoconstrictor Drugs—These drugs constrict the blood vessels thus slowing absorption This so called physiologic or chemical tourniquet effect at the site of injection prevents rapid rises in the blood levels of drugs prolongs the duration of anesthesia and produces a relatively bloodless field at the site of injection Therefore when near maximum dosages of local anesthetic agents are to be employed particularly if the area to be injected is highly vascular it is usually efficacious to incorporate a vasoconstrictor drug in the anesthetic solution

TABLE VII

CLASSIFICATION OF COMMONLY USED LOCAL ANESTHETIC AGENTS ACCORDING TO CHEMICAL STRUCTURE

Chemical Group	*Local Anesthetic Drug*
Para amino-benzoic acid esters	Novocain (procaine) Pontocaine (tetracaine) Monocaine Benzocaine Butesin Butyn
Benzoic acid esters	cocaine Metycaine (piperocaine)
Para-ethoxy benzoic acid esters	Intracaine (diethoxin)
Beta diethyl amino ethyl amide	Nupercaine (percaine dibucaine)
w diethylaminoacet 2 6-xylidide hydrochloride	Xylocaine (lidocaine)
1-cyclohexylamino-2-propyl benzoate hydrochloride	Cyclaine (hexylcaine)

Choose the Local Anesthetic Agent Carefully—If the patient is known to have had a systemic toxic reaction to one specific local anesthetic and a general anesthetic is contraindicated a local anesthetic drug of another chemical group may in most instances be used *without producing a reaction* For example Pontocaine Monocaine and Novocain are para amino benzoic acid esters cocaine and Metycaine are benzoic acid esters Nupercaine is a quinoline derivative and Xylocaine is a *basic amid* (see *Table VII page 20*) Therefore if a patient shows a reaction to a drug in one of the above four mentioned chemical groups a drug in another chemical group may conceivably be injected without causing a reaction for example if a reaction to Novocain occurs Xylocaine in most instances may be used without an untoward reaction

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However the following are some practical reasons why barbiturates are not given for this supposedly protective effect prior to all *nerve block analgesia at the Mason Clinic*

TREATMENT

The signs and symptoms of an overdose of a vasoconstrictor drug may resemble the initial stage, i.e., stimulation stage, of a toxic systemic reaction to a local anesthetic agent (see Chapter 3, page 38). However, when the reaction is caused by the vasoconstrictor drug, the blood pressure and pulse rate are increased and remain elevated, but when the reaction is caused by the local anesthetic agent, hypotension may rapidly follow the initial rise in blood pressure and pulse. Therefore, frequent checks of the blood pressure and pulse will usually reveal the cause of the reaction. Haphazard routine treatment of all reactions is to be condemned and may harm rather than help the patient.

Active treatment of systemic toxic reactions from a high blood level of a drug is aimed at (1) stopping the signs and symptoms of overstimulation of the central nervous system, (2) supplying oxygen to combat tissue hypoxia which is a result of the increased cell metabolism caused by overstimulation, (3) correcting the central depression of the cortex and vital medullary centers which follows overstimulation, (4) reversing the peripheral cardiovascular collapse, and (5) re-establishing circulation by direct manual systole in cases of cardiac arrest (standstill) or fibrillation (see Table VIII, page 24, and Table IX, page 24).

As noted previously, the onset may be immediate or delayed. In any case, the treatment is the same, and rapid institution of therapy is essential if life is to be salvaged. This is singularly true in the case of immediate onset, as well as in those rare cases of "clinical anaphylactic shock" from a true allergy, to be discussed in Chapter 2, page 35.

When a reaction begins, the physician too

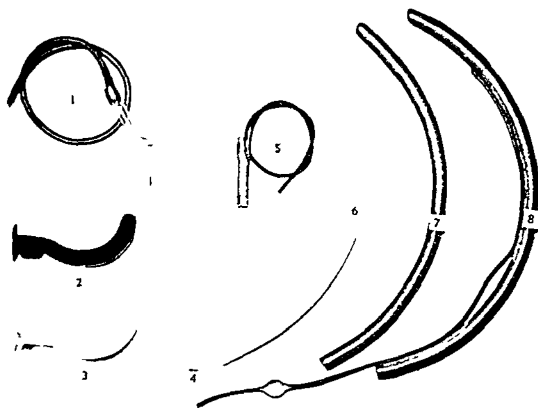


Figure 3. (1) Suction catheter with Y adapter so that intermittent suction may be applied (constant suction may damage the mucosa of the nose, pharynx and trachea). (2) Guedel oral airway. (3) Berman plastic airway. (4) Sander's inflatable cuff for an endotracheal tube. (5) Guedel cuff for endotracheal tube. (6) plastic endotracheal tube. (7) rubber endotracheal tube. (8) rubber endotracheal tube with inflatable cuff attached.

cases an area of reaction up to two inches in diameter developed Despite this degree of skin reaction the operations were carried out with procaine as the local anesthetic In no case was there any suggestion of an allergic or anaphylactic reaction or any increased tendency to the early type twitching reaction None of the eleven patients in our series who had a convulsive type of reaction had a positive skin reaction in fact in each of these cases there was no reaction to the intradermal injection of procaine hydrochloride Because of this lack of relationship between the intradermal reaction to procaine hydrochloride and the patient's general reaction to it these intradermal tests have been discontinued as a routine procedure Sadove[3] also feels that skin tests are of little use

The administration of lozenges containing the local anesthetic agent as a means of determining whether or not a patient will have a systemic reaction to the topical application of that agent has not proven to be satisfactory In one case reported recently [5] death followed the topical application of Pontocaine although no reaction occurred following the use of a lozenge containing Pontocaine hydrochloride The writer *attributed the death to an* allergy ("sensitivity") to the drug The designation *of this death as due to an allergic reaction* may be questioned the letter makes no mention as to the amount of Pontocaine contained in the lozenge the concentration of the Pontocaine applied topically or the exact amount of Pontocaine used It appears that the amount of Pontocaine sprayed into the pharynx was estimated rather than accurately measured—the author merely stated that 0 5 to 1 cc was applied Is it not likely that the reaction was of a high blood level type rather than an allergic one?

We have concluded from the foregoing as well as from our own experience at the Mason Clinic that tests to ascertain whether or not a patient will have a high blood level type of systemic toxic reaction or an allergic reaction to a local anesthetic agent prior to infiltration are impractical First, they are time consuming and second they are not conclusive[7, 10] On the other hand Adriani in closing the discussion of a paper states "In answering Dr Woodson's question regarding the use of tests some pharmacologists feel that the intradermal test is worthless I feel if one encounters a patient with a positive test, it is well to avoid the use of the drug in him"

Patch tests are of value in establishing whether or not a patient will show a skin *allergy (contact dermatitis)* to a *local* anesthetic agent (see Chapter 14 page 131) Nevertheless at the Mason Clinic the results of patch tests employed prior to the parenteral use of local anesthetic agents have not proven their worth in determining which patients will have a systemic toxic reaction However Moore[64] states "I believe that the markedly allergic individual at least should have a patch test before he is referred for bronchoscopy Even though the patch test is not 100% reliable as Thomas and Fenton have pointed out, it is an additional safeguard and will pick up some of these cases [reactions to anesthetic agents applied topically to the mucous membrane] before the anesthetic is administered" It should be emphasized here *that systemic reactions to local anesthetic* drugs applied to the mucous membranes of *the nose mouth pharynx larynx and trachea* are *not* in the greater number of instances an allergic type of systemic reaction but due to rapid absorption of highly concentrated solutions and are a high blood level type of reaction

Avoid Spreading Agents —Spreading agents such as hyaluronidase should not be used unless specifically indicated When large volumes of a local anesthetic solution are to be injected and/or they are to be placed in highly vascular areas hyaluronidase is usually contraindicated

Efocaine and Local Anesthetic Drugs in Oil —A high blood level type of reaction to *the local anesthetic drug in these mixtures* may result when large quantities are employed

Treatment of systemic toxic reactions depends on the signs and symptoms exhibited and these should be treated as they occur.

Treatment of Cortical Signs and Symptoms.—When the signs and symptoms of stimulation of the cortex, i.e., anxiety, disorientation, incoherent speech, etc., appear, *oxygen* by bag and mask should be immediately administered and intravenous fluids started. These should be the first steps in the treatment of any systemic reaction to a local anesthetic agent with or without a vasoconstrictor agent. It seems reasonable to assume that when the cortex and medulla are stimulated, the metabolic rate of their cells is greatly increased and therefore the oxygen demand is increased many fold. If the oxygen requirement of these cells is not met, it follows that hypoxia of the cells with a probable increase in CO_2 concentrations ensues and this in itself may result in convulsions, cardiovascular collapse and death. This premise has been experimentally substantiated by two separate investigations.[41,66] Tainter and Throndson[66] have shown in cats that when artificial respiration is maintained during the intravenous injection of Novocain the animal will tolerate two to ten times the amount of the drug which normally causes death. They emphasize that while the respiratory center is tem

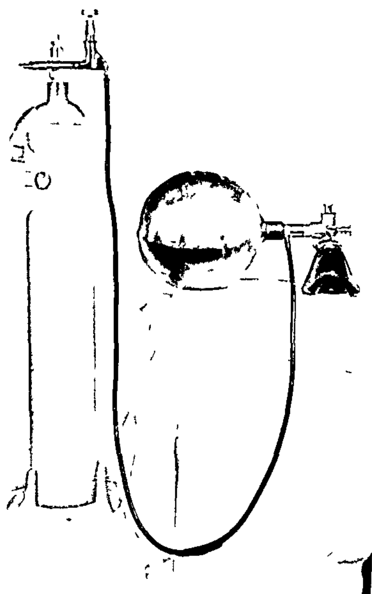


Figure 4. Minimal equipment for oxygen therapy, i.e., simple reducing valve, bag and mask.

commonly gives an intravenous barbiturate and *stimulating drugs irrespective of the* signs and symptoms. Overtreatment is to be avoided and analeptic drugs such as caffeine sodium benzoate, Coramine, Metrazol, picrotoxin, etc., *are not to be used as they merely accentuate the overstimulation and add to the depression of medullary centers*.[63] Likewise, *the indiscriminate use of barbiturates may be equally disastrous* because they may deepen the depression which follows overstimulation. Barbiturates should be used only in those cases in which convulsions are not controlled by the administration of oxygen, and then only short-acting ones should be given in small doses sufficient to stop the convulsion without producing apnea.

TABLE VIII

SUMMARY OF ACTIVE TREATMENT OF SYSTEMIC TOXIC REACTIONS FROM A HIGH BLOOD LEVEL OF LOCAL ANESTHETIC AGENT

While the following measures apply specifically to a toxic reaction from a high blood level of a local anesthetic agent, they are general principles of resuscitation and are applicable to any *reaction which may progress to shock*.

I. **Be Sure the Airway is Clear.**—If the patient becomes unconscious, establish a clear airway with an *oralpharyngeal airway* or preferably an *endotracheal tube* (Figure 3, page 23). If a patient vomits, the vomitus must be cleaned from the pharynx. If aspiration occurs, *tracheal suction* through an endotracheal tube or preferably by *bronchoscopy* should be performed. When a bronchoscope is used, it should have a sidearm so that oxygen may be insufflated into the lungs while the bronchoscopy is being done.

II. **Administer Oxygen.**—Oxygen administration should be performed with a *bag* and *mask* apparatus (Figure 4, page 25). The apparatus should be set up so that positive pressure may be used if it should become necessary to give artificial respiration to an apneic patient.

III. **Start Intravenous Fluids.**—This is an essential part of the initial treatment of a reaction and it should be done when the first signs of a reaction occur because *it assures the physician of a means of intravenous administration* of drugs even if the reaction progresses to cardiovascular collapse.

IV. **Stop Convulsions.**—This may be accomplished by intravenous injections of small amounts of a short acting barbiturate at one half to one minute intervals. The author prefers sodium Pentothal in 50 mg. doses. *DO NOT GIVE A BARBITURATE UNLESS CONVULSIONS OCCUR AND ARE PERSISTENT. IT MERELY ADDS TO THE POST STIMULATION DEPRESSION.*

V. **Raise the Blood Pressure.**—When peripheral vascular collapse starts, immediate steps to raise the blood pressure to approximately the preoperative level must be taken—use vasoconstrictor.

VI. **Institute Manual Systole.**—If cardiac arrest or fibrillation *occurs, manual systole (cardiac massage)* must be considered and rapidly instituted.

TABLE IX

EQUIPMENT NECESSARY FOR THE IMMEDIATE TREATMENT OF THE COMPLICATIONS OF REGIONAL BLOCKS

Oxygen Apparatus.—This may consist of nothing more than a bag and mask with a simple reducing needle valve for the oxygen tank, or it may be an anesthetic machine (Figure 4, page 25).

Suction Apparatus.—If *vomiting or excessive secretions* occur, it is important to clear the airway. Any type of adequate suction is satisfactory.

Airways.—Oral airways and endotracheal tubes should be available (Figure 3, page 23).

Laryngoscope and Bronchoscope.—Any type of a *Jackson or Macintosh laryngoscope will suffice if the* doctor can use it effectively. Laryngoscopy or *bronchoscopy* and intubation are often necessary to clear aspirated material from the tracheobronchial tree and to establish an unobstructed airway (Figure 5, page 26).

Intravenous Apparatus.—Intravenous fluids for supportive therapy should be readily available. Syringes to administer vasoconstrictor drugs and barbiturates should be on hand.

Instruments for Thoracotomy.—Instruments necessary to perform a thoracotomy, including a rib retractor, should always be sterile and ready for immediate use in *case cardiac arrest or ventricular fibrillation should occur*.

Cardiac Defibrillator.—If the heart is fibrillating, it must be brought to arrest before normal rhythm can be re-established.

Drugs.—The following drugs should be readily available: vasoconstrictor drugs and the barbiturates for treating hypotension and convulsions, respectively; Benadryl for *treating* allergic reactions; and calcium chloride, potassium chloride, and Pronestyl for treating cardiac failure.

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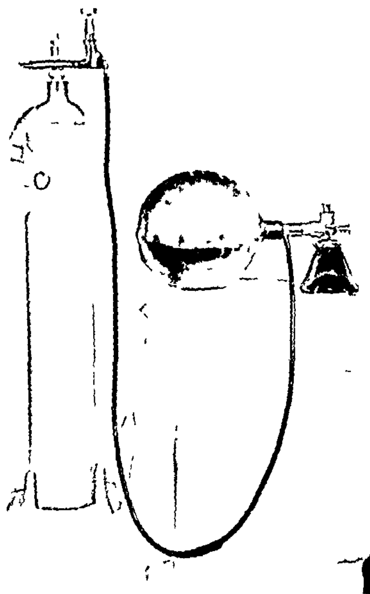


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able to the pulmonary circulation by artificial respiration It must be remembered that respirations per se are not necessary to life except as a means to supply oxygen to the alveoli of the lungs so that it may be absorbed by the pulmonary circulation In most instances as pointed out previously respiratory depression precedes circulatory collapse Cardiovascular collapse will not usually occur if when respirations become inadequate to supply sufficient oxygen to maintain normal physiological function of the body oxygen is supplied by artificial means until the local anesthetic agent's action on the respiratory center is dissipated and spontaneous respirations return

It has been the experience at the Mason Clinic, that, in the greater number of cases oxygen alone has avoided convulsions or corrected them without the use of barbiturates (see page 16) However if the patient has more than one seizure small doses (50 mg) of a short acting barbiturate, usually sodium Pentothal (thiopental) are given at one half minute intervals until the convulsions are controlled Larger doses of the short acting barbiturates given rapidly may stop the convulsions sooner but they also add to the depression of the medullary centers resulting in prolonged apnea

Treatment of Respiratory Signs and Symptoms—The early sign of stimulation of the respiratory center of the medulla by local anesthetic agents is usually only increased respiration—often very transient seldom noted and requiring no treatment On the other hand depression of the respiratory center from overstimulation by these drugs results in respiratory failure Respiratory failure evidenced by alteration of respiratory rhythm and rate should be treated immediately by administration of oxygen by bag and mask If the patient should vomit the airway must be cleared of vomitus prior to giving oxygen by bag and mask If apnea occurs then artificial respiration must be instituted Here again although the etiology differs the treatment of respiratory depression from a systemic toxic reaction to an overdosage of a local anesthetic agent is the same as that following oxygen want from spinal or epidural block (see Chapter 17 page 162 for specific therapy)

Let us emphasize that drugs which normally stimulate the respiratory center such as Coramine Metrazol or picrotoxin are contraindicated since the respiratory depression is due to previous overstimulation from the anesthetic drug Under these circumstances such drugs would only add to the depression of the respiratory center[6] Likewise the routine use of a barbiturate whenever it appears that a toxic systemic reaction has occurred is to be condemned since the depressing action of the barbiturates on the central nervous system would only further depress the respiratory center (see Chapter 5 Case 2 of Adriani page 51)

The author has seen respiratory failure occur a number of times as the *only* complication of a high blood level of a local anesthetic drug (namely Pontocaine) and in these cases artificial respiration using 100% oxygen was the only treatment necessary Thus since it is not safe to assume that cardiovascular collapse automatically accompanies precedes or follows respiratory failure the blood pressure and pulse should be checked before aiming treatment at the cardiovascular system rather than proceeding on the basis of assumption only

Treatment of Cardiovascular Signs and Symptoms—The early signs and symptoms of cardiovascular involvement are hypertension increase in pulse rate and occasionally cardiac irregularities They are usually transient reflecting a stimulation of the cardiovascular center in the medulla No special treatment is required other than oxygen therapy and the starting of intravenous fluids

The signs and symptoms of depression of the cardiovascular center in the medulla and/or the myocardium per se may follow those of stimulation or they may appear as the only signs of a reaction to a local anesthetic drug The treatment of these signs and symptoms of the depression stage is aimed at the hypotension the depressed myocardium and cardiac failure if it occurs As with cor

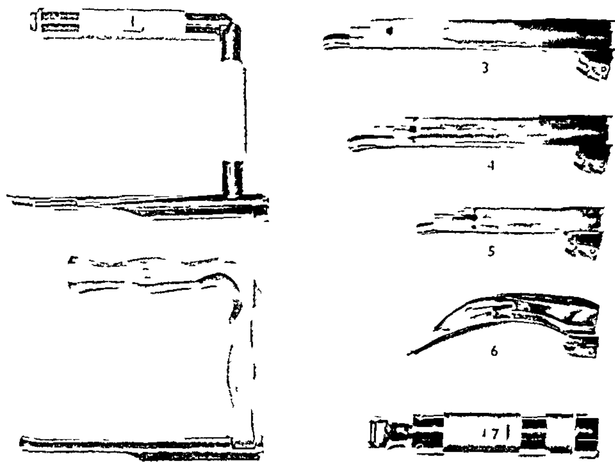


Figure 5 Laryngoscopes (1) Eversole (2) Jackson (3) large Guedel blade (4) medium Guedel blade (5) child Guedel blade (6) Macintosh blade (7) handle for detachable (hook on) blades

porarily paralyzed the artificial respiration assures oxygenation of the body[66] The work of Steinhaus[43] also supports this theory Having shown experimentally that cardiovascular collapse due to respiratory paralysis can usually be prevented by artificial respiration he concludes "Since failure of respiration occurs more readily in local anesthetic intoxication than does failure of cardiovascular function the maintenance of adequate ventilation is of primary importance even though the reaction may appear to be the result of acute cardio vascular collapse"

As noted previously the signs of minor stimulation of the brain often announce a more serious type of reaction Therefore when they occur and while oxygen is being administered a careful check of the blood pressure pulse and respiration must be maintained and drugs such as short acting barbiturates and vasoconstrictors should be readied for immediate injection in case the reaction progresses Likewise suction endotracheal tubes laryngoscopes and bronchoscopes should be on hand should the patient vomit and aspirate and/or become apneic If the patient vomits the airway must be cleared by tracheal suction and if aspiration takes place bronchoscopy may be necessary to clear the trachea and bronchi of food particles

If the minor signs of cerebral stimulation progress to convulsions which are always accompanied by periods of apnea or if convulsion is the initial indication of the reaction oxygen still is the first therapeutic agent to use This can not be too strongly emphasized If the patient is apneic either with or without convulsions the oxygen must be made avail

patient sits on the cart or operating table. This is done so that if the patient feels faint or should faint he may be placed immediately in the recumbent position.

It is interesting to note that Macintosh and Mushin[88] when discussing fainting as a complication of brachial plexus block report that fainting had " occurred only when the injection was given while the patient was sitting up. For this reason we recommend that the brachial plexus should be injected with the patient lying down."

Seldom if ever does loss of consciousness of this nature require treatment for hypotension. Placing the patient in the supine position usually suffices.

Rules To Be Remembered When Performing Regional Blocks.—*Carefully observe the patient for at least one half hour following the completion of the injection, oral administration or topical application of local anesthetic solutions.* Some reactions, particularly the mild ones, may be judged by the inexperienced physician to be hysterical in nature and not due to the drug or drugs. Should the reaction progress unobserved, death may occur before treatment can be instituted. When a patient who has been restless and difficult to block suddenly becomes quiet, the physician should not draw a sigh of relief believing that at last the patient has become cooperative. On the contrary, the physician should immediately become suspicious that a reaction is taking place and make a positive diagnosis without delay.

Objectively evaluate any type of reaction no matter how mild. Do not treat a reaction without making a definite diagnosis; give only the necessary indicated therapy.

Be prepared to treat any type of reaction. Preparedness to treat all reactions, be they mild and transient in nature or severe with convulsions and/or respiratory collapse and/or cardiovascular collapse, is essential wherever regional block procedures are performed.

Do not overtreat or undertreat the reaction. Some reactions require no treatment; on the other hand, intensive therapy and even manual systole may be necessary to save the patient's life. Either stimulation or depression of the central nervous system may predominate so that when treatment is instituted it must be "tailored" to the individual signs and symptoms—not administered in "shotgun" doses.

Tell the patient if he has a reaction. When a reaction to a drug occurs, the patient should be informed of the fact, told the drug or drugs which were employed and the method of administration so that at a later date he may pass this information on to any physician who might choose to give him a local anesthetic.

Do not exceed the recommended maximum dosage of the local anesthetic drugs (see Table VI, page 12). It should be remembered that reactions with an immediate onset following the use of a relatively large amount of a local anesthetic drug are most often seen following the *application of local anesthetic agents* to the mucous membranes of the nose or mouth, pharynx, larynx, trachea or urethra.[9, 10, 23, 69, 74]

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the lung as proved by subsequent check of the drug's blood level

It has been our custom when performing topical anesthesia to allow only a measured amount of the local anesthetic solution to be placed at our disposal The solution is poured into a medicine glass and only this amount is employed Often the over zealous resident in an effort to please his chief will exceed the toxic dosage of a local anesthetic agent if more solution is available

Do not rely on premedications to prevent systemic toxic reactions As noted previously the protection afforded by heavy doses of barbiturates is unimpressive and for the reasons previously discussed the author does not recommend them for this purpose (see page 20)

RAPID TREATMENT OF SYSTEMIC REACTIONS FROM HIGH BLOOD LEVELS OF LOCAL ANESTHETIC AGENTS IS ESSENTIAL TO AVOID DEATHS, BUT CORRECT TREATMENT IS EQUALLY IMPORTANT INAPPROPRIATE TREATMENT MAY RESULT IN DEATH!

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CHAPTER 2

Systemic Allergic Reactions to Local Anesthetic Drugs

This chapter deals only with acute systemic allergic reactions to local anesthetic drugs which occur immediately following the use of these drugs and which are characterized by acute generalized signs and symptoms such as "clinical anaphylactic shock," generalized angioneurotic edema, etc. (see definition of *allergy*, page 7). These sequelae must be treated immediately and correctly, or death may result. Chapter 14, page 127, covers dermatitis, a chronic type of allergic reaction which may be generalized or localized and which rarely requires emergency resuscitative therapy.

ETIOLOGY

Systemic allergic reactions to a local anesthetic drug used parenterally occur infrequently and in all probability constitute less than two percent of systemic toxic reactions.[1,4,15] Sadove *et al*[1] state: "Only after errors of judgment in quantity and concentration of the drug and technical faults of administration have been ruled out is it permissible to postulate any one of these states [allergy]." It must always be remembered that when *large quantities* or *high concentrations* of local anesthetic drugs are administered either topically or by injection, an actual or relative ("hyperergic") high blood level reaction, not an allergic one, is the more probable type of systemic reaction. A reaction to a drug may be termed *allergic* with certainty only if small or infinitesimal doses have been administered (see Chapter 1, page 8).

Recently Criep and Ribeiro[5] reported three deaths which they felt indicated that "allergic" reactions to Novocain (procaine) hydrochloride were more frequent than stated above. However, Ryan and Shouldice[6] and Davis and Bryce-Smith[7] question their interpretation of the cause of death in these cases, pointing out that their use of Novocain topically is somewhat surprising and that the more common explanation of the deaths has been overlooked. It is known that Novocain must be used in exceptionally high concentrations to be effective topically.[8] Ryan and Shouldice[6] further point out that in over 9,500 hernia operations using Novocain hydrochloride they have had no evidence of the type of "allergic" responses cited by Criep and Ribeiro.[5]

The signs and symptoms of an allergic systemic reaction reflect an attempt by the body to remove a noxious agent; therefore, even though the allergy results in symptoms of severe illness, it is a purposeful one of protection.[9] The intensity of the reaction is often out of proportion to the amount of the drug injected. *The cause of an allergic reaction is unknown* and Vaughan and Black[9] state: "Whatever makes a person allergic (and this we do not know as yet) makes one or another or all shock tissues abnormally responsive, so responsive that they over-act." The term "shock tissues" or "shock organs" was coined by Doerr to indicate the organs or tissues in which the allergic manifestation appears, i.e., in one person the skin may be the site of reactions, in another the bronchi, in still another the intestinal tract, etc.[9] Vaughan and Black[9] also point out that if the allergenic substance, the noxious agent, gains entrance to the body via a parenteral injection or absorp-

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and even death if given too rapidly.[7, 8, 9] Warrington *et al.*[14] do not share this opinion concerning dosage. They feel it is safe to administer 100 mg. doses of Benadryl over a 2 to 3 minute period in adults and have done so to relieve postoperative nausea and vomiting with no ill effects. They also have given as much as 400 mg. by intravenous drip over a 30 to 45 minute period with no untoward effects. Nevertheless, a dose larger than 50 mg. intravenously does not seem warranted in treating allergic responses to local anesthetic drugs because deaths have been reported from larger doses, particularly in children, and the increase in dosage does not seem to increase the drug's effectiveness greatly in alleviating the signs and symptoms of an allergic reaction.

Adrenalin (1:1000) 0.3 cc. (5 minims) may be given intramuscularly and repeated at 15 minute intervals to correct these problems. Ephedrine sulfate 50 mg. (1 cc.) may also be effective given intramuscularly. Nevertheless, it is the author's experience that Benadryl is preferable to either of these two drugs since in most instances side reactions, i.e., tachycardia, etc., often seen with Adrenalin and ephedrine sulfate are rarely brought about by the antihistaminic drugs.

Intravenous or intramuscular calcium gluconate 1 to 2 gm. (5 to 10 cc. of a 20% solution) may prove valuable to control pruritis. It should be remembered that if calcium chloride is used instead of the gluconate, it must not be given intramuscularly or subcutaneously because of its necrotizing action. Lotions containing calamine and phenols may alleviate the itching but they are not as effective as the antihistaminic drugs.

Asthmatic Breathing.—This may be relieved by the administration of oxygen only or oxygen-helium mixtures. If this treatment does not help within a relatively short period of time, drug therapy should be instituted. A 50 mg. dose of Benadryl given intravenously may suffice to stop the attack but Adrenalin (epinephrine) and/or Aminophylline (theophylline with ethylenediamine) are usually more effective. Adrenalin (1:1000) 0.3 cc. (5 minims) may be given intramuscularly and repeated at 15 minute intervals, or if the attack is persistent and seems to be intractable, a dose of 1 cc. of a 1:10,000 concentration may be given *slowly* intravenously. However, Aminophylline 7½ gr. (450 mg.) given *slowly* intravenously in the adult is the author's drug of choice—it often produces dramatic relief in asthmatic conditions refractory to Adrenalin.[10] Rapid injection may produce unpleasant side effects such as flushing, palpitation, and at times even syncope and collapse. A similar dose of Aminophylline may be repeated in 15 to 30 minutes. If it is more convenient to use the intravenous drip method, 15 gr. (900 mg.) may be dissolved in 1000 cc. of fluid, usually 5% dextrose in distilled water, and dripped rapidly at first until the asthmatic breathing ceases, then slowed to a maintenance drip.

Hypotension.—A fall in blood pressure with an associated hypoxia may occur by itself in allergic conditions or it may accompany "clinical anaphylactic shock." It should be corrected immediately by intravenously administered vasoconstrictor drugs (see Chapter 15, page 147 for specific therapy).

Laryngeal Edema.—Laryngeal edema may result in the occasional case, and if severe, intubation or tracheotomy may be necessary to obviate respiratory obstruction and prevent oxygen want. However, if angioneurotic edema is treated immediately, laryngeal edema is less likely to occur.

Clinical Anaphylactic Shock.—Shock of this type seldom occurs, but when it does, treatment is the same as for a severe systemic reaction from a high blood level of a local anesthetic drug (see Chapter 1, pages 23 to 28 for specific therapy). In addition, if an allergy to a local anesthetic drug is thought to have caused this reaction, antihistaminic drugs intravenously in the doses noted above may prove valuable. Death is not unusual in this type of reaction, but with the rapid institution of therapy some fatalities may be avoided. Treatment should include artificial respiration with high concentrations of oxygen, intravenous fluids, vasoconstrictor agents, and manual systole. In these cases hypoxia is probably responsible for the convulsions,

tion from the respiratory or gastric mucosa the clinical response may be seen in any or all of the shock tissues and not localized to the point of entrance as it is with a contact type dermatitis (see Chapter 14 page 127)

SIGNS AND SYMPTOMS

The signs and symptoms of allergy to a local anesthetic drug requiring immediate therapy are reflected by one or more of the following *generalized angioneurotic edema urticaria pruritis hypotension joint pains asthmatic breathing nausea and vomiting and occasionally "clinical anaphylactic shock"* [1 9 10 11] It must be remembered that when angioneurotic edema occurs it may involve the larynx and its associated structures with resultant severe respiratory obstruction In DeBold and Fox's [11] own series of over 1 600 intravenous procaine treatments in 300 patients there were only three allergic reactions and these were characterized by nausea wheezing and shortness of breath" To date allergic reactions of this nature have not been seen at the Mason Clinic

The administration of infinitesimal amounts of local anesthetic solutions e g raising of a skin wheal prior to insertion of large needles may on rare occasions be followed by "clinical anaphylactic shock Shock of this type is usually characterized by a sudden cardiovascular and respiratory collapse which if not adequately treated may be fatal in 1 to 5 minutes Equally infrequent is a systemic allergic reaction resulting from the small dosages of local anesthetic drugs required for spinal anesthesia However Nowill [17] reports a case of convulsion following the administration of a spinal anesthetic of 17 mg of Pontocaine He and Stephen [18] believe that this reaction signified a true allergy to the drug

PROPHYLAXIS

Prophylaxis is at best not very satisfactory in allergy It consists of taking an adequate history and the performance of a number of time-consuming tests the results of which are often not significant [9 16]

History —If the history is adequately taken it will prove to be the most valuable source of information but as Vaughan and Black [9] point out, to be of value the allergic history must be painstakingly obtained" An adequate history does not mean just asking the patient whether he has had a local anesthetic agent injected previously and whether or not any untoward effect had followed the circumstances under which the reaction occurred must be ascertained as well as the drug or drugs used This is most important, as unpleasant reactions are often caused by high concentrations of vasoconstrictor agents In addition to a personal history, reactions in other members of the family should be inquired into such evidence might reveal an important clue since heredity often plays an important part in allergy [9]

Skin Wheals, Lozenge Patch and Mucous Membrane Tests — Intradermal skin wheal tests and tests applied to the mucous membranes may occasionally prove whether or not the patient who is known to be markedly allergic to other substances will have an allergic reaction to the injection or topical application of a local anesthetic agent But since these tests are time consuming and since serious *allergic* reactions are very infrequent the practicality of such tests prior to performing local analgesia for an operative procedure or for diagnostic and therapeutic blocks is questionable (see Chapter 1 page 21)

TREATMENT

Treatment consists of the following specific therapy of the signs and symptoms

Angioneurotic Edema, Wheals (urticaria) and Pruritis (itching) —These are best treated by an antihistaminic drug such as Benadryl (diphenhydramine) orally and intravenously The dosage for Benadryl is 50 mg administered either orally or slowly intravenously This dosage may be repeated at 3 to 4 hour intervals as needed Larger intravenous doses of Benadryl i e 100 mg should be used with caution as they are seldom of more therapeutic benefit and may result in nausea and vomiting convulsions cardiac irregularities

CHAPTER 3

Systemic Reactions to Vasoconstrictor Drugs Used to Prolong the Effect of Local Anesthetic Drugs

As with the local anesthetic agents, two main types of toxic reactions to vasoconstrictor drugs are usually recognized, i.e., *systemic* and *local* toxic reactions. Local toxic tissue reactions from these drugs are discussed in Chapter 10, page 96.

Vasoconstrictor drugs are used in regional block procedures for primarily three purposes: (1) to prolong the anesthetic effect, (2) to prevent hypotension, and (3) to correct hypotension. This chapter deals principally with the use of Adrenalin (epinephrine) to prolong the effect of local anesthetic drugs. However, much of the discussion concerning systemic toxic reactions to Adrenalin applies to any vasoconstrictor drug regardless of the purpose for which the drug was employed.

ETIOLOGY

Systemic toxic reactions to vasoconstrictor drugs may be the result of: (1) an allergy to the drug, or (2) a high blood level of the drug. It must be remembered when classifying systemic reactions that the amount of a drug tolerated by the majority of patients may be an overdosage in the minority of individuals, i.e., hyperergy (see Chapter 1, page 8). Smaller doses of the drug may be used in this minority without untoward effects. Therefore, when a systemic reaction to a vasoconstrictor drug occurs, it must not be assumed that it is due to an allergy, for in most instances the reaction reflects an overdose.

Allergy.—Allergic systemic toxic reactions to a drug have been defined as characterized by dermatitis, angioneurotic edema, urticaria, pruritus, hypotension, asthmatic breathing, or "clinical anaphylactic shock." In over 8000 regional nerve block procedures (spinal and epidural blocks not included) performed at the Mason Clinic in which Adrenalin (epinephrine) has been used, no such reaction has been observed. Bonica[1] who has performed an equally large number of blocks, confirms the absence of this type of reaction from vasoconstrictor drugs. The literature reviewed is devoid of reports of such allergic reactions with the following two exceptions. Halle[2] has observed spasmodic sneezing (from 50 to 100 times) in a number of cases following introduction of Adrenalin into the nasal duct, either with a tampon or a spray, and this complication might be considered an allergic type of reaction. Gordonoff and Steube[3] have suggested that death or severe reactions after Novocain-Adrenalin injections in two of their cases may be due to an allergic reaction to Adrenalin, i.e., "clinical anaphylactic shock" as defined by Vaughan and Black[4] in Chapter 1, page 7. However, the 2 cases of Gordonoff and Steube[3] are not convincing and are open to question. One patient was known to have a very labile cardiovascular system, and in the other case the Novocain-Adrenalin solution was given orally.

High Blood Level.—While allergic reactions to vasoconstrictor drugs are rare, reactions to a high blood level of these drugs are not infrequent when the usual recommended optimum dosages of these drugs are used in local anesthetic solutions. The blood level which a vasoconstrictor *agent* attains is directly related to the rates of absorption, detoxification

therefore the convulsions should not be treated with an intravenous barbiturate initially since oxygenation alone may eliminate the convulsions If a barbiturate is used inadvisedly in this type of case, it adds further to the cerebral and respiratory depression of the patient Should cardiac failure occur manual systole must be performed immediately (see Chapter 7 page 76)

COMMENT

Acute Delayed Systemic Allergic Reactions —Two cases are recorded which were labeled as acute systemic allergic reactions and which did not occur for two or more hours after the local anesthetic drug gained entrance to the body [19] This type of an acute systemic allergic reaction if it is caused by an allergy is quite unusual because of the delayed onset

The first case was that of a three year old child who ingested eight Nupercaine (dibucaine) lozenges i e a total dosage of 8 5 mg of the drug Two hours after taking the lozenges she was comatose and cyanotic A soft non pitting edema appeared on the face The blood pressure was 60/40 and the pulse was slow She was breathing four to five times a minute and her respirations were irregular and shallow Subsequently this child died and autopsy revealed no apparent cause of death The author stated that this amount of the drug would not ordinarily cause symptoms of importance in a child of this size and concluded that the soft non pitting edema was apparently a sign of an allergic reaction

The second case also cited in the report of the above case occurred following the administration of a spinal anesthesia with Pontocaine (tetracaine hydrochloride) The patient also became comatose and exhibited generalized soft non pitting edema similar to that noted in the first case No other information concerning the dosage or the signs and symptoms in this case was made available by the article According to the author the second case "recovered with the help of parenteral cortisone after several days of a critical illness"

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PROPHYLAXIS

In order to avoid a systemic toxic reaction to a vasoconstrictor drug, the following pertinent points must be considered and evaluated when adding such a drug to a local anesthetic solution.

Indications for Use.—Unless vasoconstrictor drugs are employed for a specific purpose, i.e., prolongation of anesthesia and/or reduction of the rate of absorption of the local anesthetic solution and/or a bloodless field, they should be omitted from the local anesthetic solution. Adriani states: "I avoid the use of Adrenalin in solutions of local anesthetic drugs because the stimulation caused by epinephrine is often confused with the prodromal signs of a toxic reaction."

At the Mason Clinic a large percentage of the patients who are to have diagnostic and therapeutic blocks are treated as outpatients, and it has become routine to omit the vasoconstrictor drug in these cases because systemic toxic reactions from a high blood level of these drugs are frequent even when less than the optimal recommended dose is used.

Drug of Choice.—Many vasoconstrictor drugs are available, but from the time Braun[8] advocated the use of Adrenalin to date, only Adrenalin (epinephrine) and to a lesser extent Cobefrin (corbasil) and Neosynephrine (phenylephrine) have been found useful. The powerful peripheral vasoconstrictor drugs, i.e., Levophed (levarterenol), etc., *should not* be used for infiltration analgesia until they have undergone more extensive experimental study (see Chapter 15, page 150).

Optimum Concentrations.—Bieter[9] and Leser[10] have shown that for the best results Adrenalin should be used in a final concentration of 1:200,000, i.e., 0.1 cc. of 1:1000 Adrenalin per 20 cc. of a local anesthetic solution. The optimal concentration of Cobefrin is 1:40,000, while that for Neosynephrine is 1:20,000.

At the Mason Clinic, Adrenalin is the vasoconstrictor of choice for use in local anesthetic blocking solutions. It has been our experience that exceeding the dose of 0.20 to 0.25 cc. of 1:1000 Adrenalin at any one time raises the incidence of reactions even in those surgical patients who have received adequate premedication. Therefore, we never exceed a dosage of 0.25 cc. of a 1:1000 solution of Adrenalin even though a dose of 100 to 150 cc. of the local anesthetic solution is employed for the block. Consequently, the vasoconstrictor concentration of many of our solutions is below the optimal. However, Pontocaine is our drug of choice for regional block procedures, and even with concentrations of Adrenalin below the optimum level analgesia lasts from 4 to 6 hours. Likewise, it has been found that if more than 2 mg. of Neosynephrine are injected into the body, toxic reactions occur more frequently. Therefore, irrespective of the amount of local anesthetic solution used, the amount of Neosynephrine mixed with it never exceeds 2 mg. These dosages apply only to infiltration and nerve block procedures, as it has been found that higher dosages may be used in the subarachnoid space without causing a high blood level of the drug. This is true probably because the blood supply to the spinal cord is relatively poor and is limited to small blood vessels which are immediately constricted by the vasoconstrictor drug, thereby automatically delaying rapid absorption.

The author has had no experience with Cobefrin and cannot elaborate further on its usage. It should be noted, however, that it is an effective synthetic vasoconstrictor which is supposed to cause fewer untoward reactions than Adrenalin, i.e., less palpitation, tachycardia, pallor, tremors, etc. Bieter[9] and Hellijas and Tovell[11] recommend it highly in concentrations of 1:20,000 to 1:80,000, depending on whether 2.0 or 0.5% Novocain is employed. However, Tainter and Throndson[12] after a careful investigation of Cobefrin came to the conclusion that it did not have much to recommend it over Adrenalin and that local anesthetic solutions containing Adrenalin would be less likely to cause a fatal systemic reaction.

Method of Measuring.—The dose of vasoconstrictor drugs to be used should be meas-

and elimination of that drug and these are influenced by the following factors (1) toxicity of the drug (2) total weight of the drug injected (3) concentration of the drug in the anesthetic solution (4) vascularity of the area injected (5) inadvertent intravenous injections, (6) rapidity of onset of the vasoconstriction effect (7) rate of injection (8) the use of spreading agents (9) physical status of the patient and (10) environmental temperature It will be noted that these factors are applicable to vasoconstrictor drugs and local anesthetic agents alike and since they are adequately reviewed in a previous chapter their discussion here is superfluous (see Chapter 1 pages 9 to 16)

SIGNS AND SYMPTOMS

Allergy to Vasoconstrictor Drugs—While *local* tissue reactions from sensitization to Adrenalin have been reported (see Chapter 10 page 96) proven cases of generalized systemic allergic reaction to vasoconstrictor drugs characterized by dermatitis urticaria and pruritis must be infrequent Little concerning them appears in the literature

High Blood Level of Vasoconstrictor Drugs—Most reactions to vasoconstrictor drugs result from an overdosage with a high blood level of the drug These reactions are usually characterized in the healthy adult by palpitation, hypertension tachycardia fainting tachypnea respiratory difficulty fear anxiety restlessness apprehension headache nausea, vomiting tremor weakness pallor and coldness of the skin Of these palpitation and/or severe intolerable headache are almost pathognomonic of a high blood level of a vasoconstrictor drug and are perhaps the most frequent subjective symptoms of this type of reaction Occasionally such a systemic reaction may precipitate a cerebral hemorrhage a cardiac irregularity a coronary occlusion or may aggravate the pain of an existing angina In the psychoneurotic individual existing symptoms may be aggravated[9]

The signs and symptoms of a reaction to vasoconstrictor drugs often resemble the early stages of a toxic reaction to a local anesthetic drug i e stimulation of the central nervous system This has led many clinicians to misinterpret them and blame the local anesthetic agent for the reaction rather than the true factor—the vasoconstrictor drug At the Mason Clinic many patients have been encountered who had been told that they were "allergic" to local anesthetic agents because they had exhibited such signs and symptoms following local infiltration in a dentist's or another physician's office After careful investigation of the circumstances in which the reaction occurred it was concluded that most of these reactions thought to be caused by the local anesthetic drug were in reality due to the vasoconstrictor drugs Our conclusions have been further substantiated because we have not hesitated to block these patients with local anesthetic solutions containing either small amounts (0 2 to 0 25 cc or less) or no Adrenalin at all without complications These cases do not prove that vasoconstrictor drugs should be omitted from local anesthetic solutions but stress the necessity of using proper doses of the drugs—not overdoses

In addition to these usual signs and symptoms of toxic reactions to a vasoconstrictor drug an isolated report by Seltzer[6] has attributed convulsions to an overdosage of Adrenalin He believes the convulsions in the reported case were due to hyperventilation tetany produced by the stimulation effect of the drug on the patient's central nervous system The dose of Adrenalin administered was 5 minims of a 1 1000 solution The reaction was characterized by a rise in pulse rate from 80 to 140 a rise in blood pressure from 120 to 150 a rise in respiratory rate from 18 to 45 palpitation choking sensation dyspnea weeping panting and intermittent generalized tonic and clonic convulsions lasting from 30 to 90 seconds which recurred every 2 to 4 minutes No headache followed the episode and carpopedal spasm involuntary defecation and urination did not occur during the convulsions

PROPHYLAXIS

In order to avoid a systemic toxic reaction to a vasoconstrictor drug the following pertinent points must be considered and evaluated when adding such a drug to a local anesthetic solution

Indications for Use—Unless vasoconstrictor drugs are employed for a specific purpose i e prolongation of anesthesia and/or reduction of the rate of absorption of the local anesthetic solution and/or a bloodless field they should be omitted from the local anesthetic solution Adriani states "I avoid the use of Adrenalin in solutions of local anesthetic drugs because the stimulation caused by epinephrine is often confused with the prodromal signs of a toxic reaction"

At the Mason Clinic a large percentage of the patients who are to have diagnostic and therapeutic blocks are treated as outpatients and it has become routine to omit the vasoconstrictor drug in these cases because systemic toxic reactions from a high blood level of these drugs are frequent even when less than the optimal recommended dose is used

Drug of Choice—Many vasoconstrictor drugs are available but from the time Braun[8] advocated the use of Adrenalin to date only Adrenalin (epinephrine) and to a lesser extent Cobefrin (corbasil) and Neosynephrine (phenylephrine) have been found useful The powerful peripheral vasoconstrictor drugs i e Levophed (levarterenol) etc *should not* be used for infiltration analgesia until they have undergone more extensive experimental study (see Chapter 15 page 150)

Optimum Concentrations—Bieter[9] and Leser[10] have shown that for the best results Adrenalin should be used in a final concentration of 1 200 000 i e 0 1 cc of 1 1000 Adrenalin per 20 cc of a local anesthetic solution The optimal concentration of Cobefrin is 1 40 000 while that for Neosynephrine is 1 20 000

At the Mason Clinic Adrenalin is the vasoconstrictor of choice for use in local anesthetic blocking solutions It has been our experience that exceeding the dose of 0 20 to 0 25 cc of 1 1000 Adrenalin at any one time raises the incidence of reactions even in those surgical patients who have received adequate premedication Therefore we never exceed a dosage of 0 25 cc of a 1 1000 solution of Adrenalin even though a dose of 100 to 150 cc of the local anesthetic solution is employed for the block Consequently the vasoconstrictor concentration of many of our solutions is below the optimal However Pontocaine is our drug of choice for regional block procedures and even with concentrations of Adrenalin below the optimum level analgesia lasts from 4 to 6 hours Likewise, it has been found that if more than 2 mg of Neosynephrine are injected into the body toxic reactions occur more frequently Therefore irrespective of the amount of local anesthetic solution used the amount of Neosynephrine mixed with it never exceeds 2 mg These dosages apply only to infiltration and nerve block procedures as it has been found that higher dosages may be used in the subarachnoid space without causing a high blood level of the drug This is true probably because the blood supply to the spinal cord is relatively poor and is limited to small blood vessels which are immediately constricted by the vasoconstrictor drug thereby automatically delaying rapid absorption

The author has had no experience with Cobefrin and cannot elaborate further on its usage It should be noted however that it is an effective synthetic vasoconstrictor which is supposed to cause fewer untoward reactions than Adrenalin i e less palpitation tachycardia pallor, tremors etc Bieter[9] and Hellijas and Tovell[11] recommend it highly in concentrations of 1 20 000 to 1 80 000 depending on whether 2 0 or 0 5% Novocain is employed However Tainter and Throndson[12] after a careful investigation of Cobefrin, came to the conclusion that it did not have much to recommend it over Adrenalin and that local anesthetic solutions containing Adrenalin would be less likely to cause a fatal systemic reaction

Method of Measuring—The dose of vasoconstrictor drugs to be used should be meas

and elimination of that drug, and these are influenced by the following factors (1) toxicity of the drug (2) total weight of the drug injected (3) concentration of the drug in the anesthetic solution (4) vascularity of the area injected (5) inadvertent intravenous injections (6) rapidity of onset of the vasoconstriction effect (7) rate of injection (8) the use of spreading agents (9) physical status of the patient and (10) environmental temperature It will be noted that these factors are applicable to vasoconstrictor drugs and local anesthetic agents alike and since they are adequately reviewed in a previous chapter their discussion here is superfluous (see Chapter 1 pages 9 to 16)

SIGNS AND SYMPTOMS

Allergy to Vasoconstrictor Drugs—While *local* tissue reactions from sensitization to Adrenalin have been reported (see Chapter 10 page 96) proven cases of generalized systemic allergic reaction to vasoconstrictor drugs characterized by dermatitis urticaria and pruritis must be infrequent. Little concerning them appears in the literature

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In addition to these usual signs and symptoms of toxic reactions to a vasoconstrictor drug an isolated report by Seltzer[6] has attributed convulsions to an overdosage of Adrenalin He believes the convulsions in the reported case were due to hyperventilation tetany produced by the stimulation effect of the drug on the patient s central nervous system The dose of Adrenalin administered was 5 minims of a 1 1000 solution The reaction was characterized by a rise in pulse rate from 80 to 140 a rise in blood pressure from 120 to 150 a rise in respiratory rate from 18 to 45 palpitation choking sensation dyspnea weeping panting and intermittent generalized tonic and clonic convulsions lasting from 30 to 90 seconds which recurred every 2 to 4 minutes No headache followed the episode and carpopedal spasm involuntary defecation and urination did not occur during the convulsions

nitrite should not be administered in a closed system since a methemoglobinemia may result.[6]

Adrenolytic Drug.—Chlorpromazine (Thorazine, Largactil, etc.) has been shown to depress the pressor action of Levophed (norepinephrine) and reverse the action of Adrenalin.[14] Therefore, a small intravenous dosage (5 to 10 mg.) may be administered when the signs and symptoms of a systemic reaction from a high blood level of Adrenalin do not subside in response to other therapy. *Certainly its use should be considered if a massive dose of Adrenalin which may be fatal is injected inadvertently.* If the desired effects from the small initial injection are not obtained, additional 5 mg. dosages may be given at intervals of 15 minutes. It should be warned that a larger initial dosage of chlorpromazine (20 to 50 mg.) should not be given intravenously as it normally causes a precipitous fall in blood pressure which may respond only to a rapid intravenous drip of Levophed or Neo-synephrine.

COMMENT

Cardiac Arrhythmias.—It has been noted in Chapter 1, page 17, that irregularities in cardiac rate and rhythm may occur following the administration of local anesthetic solutions and that they may be caused by the anesthetic drug. It must, therefore, be emphasized that when epinephrine or "epinephrine-like" substances are incorporated in these solutions an irregularity may develop that may be due to the vasoconstrictor drug, not to the local anesthetic agent. The type of irregularity usually seen in these cases is a coupling of premature auricular contractions with a normal sinus beat. This is not due to any action on the A-V node or the conductive system of the heart but is caused by an irritability of the auricle which discharges just after the refractory period of the S-A node.[15]

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ured accurately with either a 2 to 3 cc syringe or a tuberculin syringe There is a wide difference in dropper shapes and outlets and the measuring of these solutions by the drop method is to be condemned unless the dropper is calibrated

Use Small Ampules —Because of deterioration small ampules of vasoconstrictor drugs should be used rather than large rubber stoppered bottles, and they should be opened as needed Adrenalin and Neosynephrine are particularly liable to decompose when exposed to air for a period of time Solutions of Adrenalin and Neosynephrine are normally clear and colorless if they are pink brownish red and/or turbid they are deteriorating and should be discarded

Avoid Prepared Local Anesthetic Solutions Containing Adrenalin —Solutions containing Adrenalin may be purchased directly from pharmaceutical houses and they usually contain relatively high concentrations of Adrenalin to prolong analgesia This type of solution in addition to causing a high number of systemic toxic reactions from the vasoconstrictor drugs may actually be responsible for the tissue irritation which can follow the use of Xylocaine and Novocain and which is apparently due to metal ions that the blocking solution releases from regional block equipment (see Chapter 10 page 94 and Chapter 11 page 114)[13]

TREATMENT

It should be again stressed that generalized systemic *allergic* reactions to vasoconstrictor drugs seldom if ever occur Therefore the treatment of systemic reactions outlined as follows, is limited to the treatment of *over dosage* of the drug

Treatment of systemic toxic reactions to vasoconstrictor drugs should fit the severity of the signs and symptoms In many instances the reaction is minor and justifies no therapy other than watchful waiting Nevertheless the patient must be carefully watched because as noted previously the early signs and symptoms of a systemic toxic reaction to a local anesthetic drug may closely resemble those of a reaction to a vasoconstrictor drug Should a severe systemic toxic reaction to either agent ensue in a patient not being carefully observed, death may result

Usually a careful check of the blood pressure pulse and respiration will give a good clue as to the type of reaction If the blood pressure is elevated and remains so and the characteristic signs and symptoms of a reaction to a vasoconstrictor drug become prominent, the following therapy should be instituted

Oxygen by Bag and Mask—If the patient is particularly apprehensive oxygen administered by bag and mask may relieve his dyspnea restlessness, palpitation and other minor complaints If on the other hand the reaction becomes severe administration of oxygen is a necessity to supply the increased oxygen demand of the body which results from over stimulation

Intravenous Barbiturates—If the administration of oxygen alone does not calm the patient small intravenous doses of short acting barbiturates i e 50 mg of sodium Pentothal (thiopental) or Nembutal (pentobarbital) at 2 minute intervals may produce enough sedation without loss of consciousness to quiet the patient and keep him comfortable until the reaction has passed Usually this type of barbiturate therapy will reduce the hypertension and thereby relieve the severe headache which is associated with high blood pressure On rare occasions it may be necessary to give these drugs continuously over a period of 2 to 3 hours and keep the patient lightly asleep

Vasodilator Drugs—Should the hypertension persist amyl nitrite by inhalation nitroglycerin 0 5 mg sublingually or intravenous Aminophylline 225 mg (3¾ gr) may be necessary to lower the blood pressure to normal limits This is important in those patients particularly the elderly ones in whom prolonged vasoconstriction may result in a coronary occlusion cerebral vascular accident cardiac dilatation or cardiac failure Amyl

from overstimulation, i.e., primary oxygen want, is correct.

Systemic Toxic Reactions to Vasoconstrictor Drugs.—Following local infiltration and peripheral nerve blocks where excessive amounts of vasoconstrictor drugs are employed in the local anesthetic solution, an increase in the metabolic processes of the body cells may result, i.e., a demand hypoxia. An identical situation may be created by a prophylactic dose of a vasoconstrictor agent given before a spinal or epidural block.

Pneumothorax.—Following inadvertent trauma to the parietal or pulmonary pleura and/or the lung parenchyma, the lung, or part of it, may collapse and oxygen want ensue. This would be classified as alveolar hypoxia.

Overflow of Solution onto Nerves Other Than Those To Be Blocked.—When performing regional block procedures, particularly those of the head and neck, it is not uncommon to block nerves other than those to be anesthetized. In most instances when local anesthetic solutions are employed, such an overflow creates no problem and requires no treatment. However, at times resuscitative measures such as artificial respiration and maintaining blood pressure must be instituted until the drug's action has worn off.

On the other hand, if overflow occurs with the use of long-acting blocking agents, i.e., neurolytic agents, a serious problem may be created which may necessitate prolonged hospitalization and additional medical care to prevent oxygen want and death. Law suits which may result from this are discussed in Chapter 12, page 119. Treatment in these cases is only symptomatic and must be carried out until the nerve regenerates, which may take six months to one year. However, occasionally the sequelae of the complication may be permanent—a fact which only time will reveal.

The nerve or nerves which may be commonly inadvertently blocked and the block of which may result in primary oxygen want from overflow of the anesthetic solution are:

The Phrenic Nerve.—The phrenic nerve is paralyzed in a relatively large number of patients receiving a brachial plexus block, or

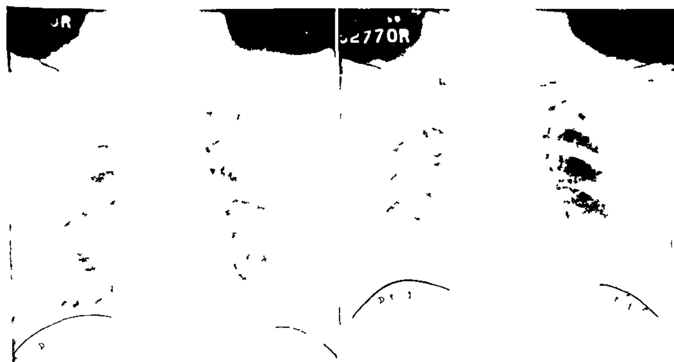


Figure 6. Bilateral phrenic block following a bilateral brachial block in this 75 year old patient presented the signs and symptoms of acute oxygen want because of the marked emphysema. His distress lasted six hours until the block dissipated itself. During this time an oxygen tent was necessary. *Left*, Chest x-ray previous to surgery. *Right*, Chest x-ray two hours postoperatively. Note the bilateral elevation of the diaphragm.

Primary Oxygen Want and Hypotension

Oxygen want and hypotension following local infiltration and peripheral nerve block are *seldom* encountered unless systemic toxic reactions to the local anesthetic drug, a systemic toxic reaction to the vasoconstrictor drug, or a pneumothorax ensues. In such cases, if the oxygen want goes untreated, a hypotension results, i.e., the oxygen want is the principal cause of the hypotension. This sequence of events is opposite to that which usually follows an inadvertent or purposely administered spinal and epidural block where in the authors' opinion the hypotension is usually primary and the oxygen want is secondary (see Table XI, page 46).

TABLE X

CLASSIFICATION OF HYPOXIA (OXYGEN WANT)
(*After Saklad*[1])

Types	*Definition*
ATMOSPHERIC HYPOXIA	reduction in the availability of oxygen to the tissue because of a diminution in the partial pressure of oxygen in the inhaled atmosphere
TIDAL *HYPOXIA*	decrease in oxygen uptake by *tissue because of a decreased respira*tory exchange
ALVEOLAR HYPOXIA	decrease in the number or efficiency of functioning alveoli
HEMOGLOBIC HYPOXIA	the total load of oxygen is reduced in proportion to the reduction in hemoglobin content, as in hemorrhage, or to a reduction in the amount of available hemoglobin, as in carbon monoxide poisoning
STAGNANT HYPOXIA	result of slowed circulation
HISTOTOXIC HYPOXIA	utilization of oxygen by the cells is interfered with
DEMAND HYPOXIA	metabolic conditions in which the bodily requirements for oxygen are elevated above *normal.*

ETIOLOGY

The types of oxygen want which may occur are found in Table X, page 42, and their definitions should be clearly in mind. The etiological factors of primary oxygen want following local infiltration and peripheral nerve block analgesia are:

Systemic Toxic Reactions to the Local Anesthetic Drug.—In this type of reaction, from a pharmacological standpoint at least, the reaction is primarily due to a depression of the respiratory center after overstimulation (*see Chapter 1, page 16*).[2] But the depressing action reported of most local anesthetic agents on the *cardiovascular system proposes* the question: Are not some of these reactions due to the direct depression of this system or perhaps to a combination of this and depression of the respiratory center in the medulla (see Chapter 1, page 16)?

The hypoxia following this type of reaction would be classified as either tidal (depression of the respiratory center) or stagnant (depression of the cardiovascular system) or a combination of the two. Although further re*search on this problem is needed,* it must be assumed for the time being that the current theory *of depression of the respiratory center*

the Accessory Nerves and Fibers of the Sympathetic Nervous System.—Blocks of all these nerves may follow an attempt to block any one of them as they emerge from the cranium via the jugular foramen where they all lie in close approximation (see Figure 7, page 41). When a unilateral block of these nerves results at this site, no problem occurs other than hoarseness and an occasional tachycardia from paralysis of the vagus nerve. The trapezius muscle and one half the tongue are also blocked at the same time but this will not entail serious consequences.

On the other hand, bilateral block of all these nerves results in complete anesthesia of the pharynx, larynx and the tongue. This may allow the tongue to fall into the pharynx and/or the vocal cords to adduct. Either or both of these may occlude the airway. This complication is classified as a tidal hypoxia.

Premedication.—Excessive premedication with barbiturates or the opiates may result in tidal hypoxia from depressed respirations.

Contributing Factors.—Often regional blocks are chosen for the patient with a poor physical status who has a chronic hypoxia from diseases such as tuberculosis (alveolar hypoxia), emphysema (tidal and alveolar hypoxia), anemia (hemoglobic hypoxia), and hyperthyroidism or hyperpyrexia (demand hypoxia). It has also been the anesthesia of choice at high altitudes (atmospheric hypoxia) and in patients with drug poisoning (histotoxic hypoxia) because of the difficulties involved in administration of inhalation anesthetics under these circumstances. When administering a regional block to a patient in any of these circumstances, it must be realized that any hypoxia caused by the anesthetic procedure itself will automatically accentuate the pre-existing chronic tissue hypoxia and may result in acute respiratory distress.

SIGNS AND SYMPTOMS

When severe, acute *primary* oxygen want develops from a systemic toxic reaction, an obstruction of the pharynx or larynx, or damage to the pulmonary parenchyma of both lungs, the cortical and respiratory signs and symptoms are the same as those seen when hypoxia is secondary to hypotension due to paralysis of portions of the sympathetic nervous system following an inadvertent or purposely administered spinal or epidural block. However, the appearance of the patient and the cardiovascular signs and symptoms differ (see Table XI, page 46).

Cortical Manifestations.—The cortical signs and symptoms of oxygen want are the same regardless of the etiology (see Chapter 17, page 160). Following local and peripheral nerve block they may progress from one to another more rapidly than following a purposely administered spinal or epidural block.

Respiratory Manifestations.—Regardless of the cause, the manifestations of respiratory insufficiency are the same (see Chapter 17, page 161).

Cardiovascular Manifestations.—When primary oxygen want develops, a rise in the blood pressure and pulse rate may be noted immediately if a recording is taken. But such a check is usually not made since requisite help is not available or the physician is nonchalant about regional block procedures, particularly local infiltration. Thus by the time a check is made, the blood pressure may still be elevated but the pulse, slow, full and bounding. This kind of situation may fool the physician, but the diagnosis should not be missed particularly where there appear other signs of *primary* oxygen want, i.e., respiratory distress and cyanosis. This type of oxygen want is the opposite of the type which develops following spinal or epidural block where the pulse is rapid, the blood pressure low, and no cyanosis develops.

Appearance of the Patient.—Early in primary oxygen want the body is deprived of oxygen but circulation is not markedly altered; therefore marked cyanosis appears and should serve as a warning to the physician that all is not well. The two exceptions to this in local infiltration and peripheral nerve block are the oxygen want which develops following the administration of a vasoconstrictor drug and that which occurs in the patient with a severe

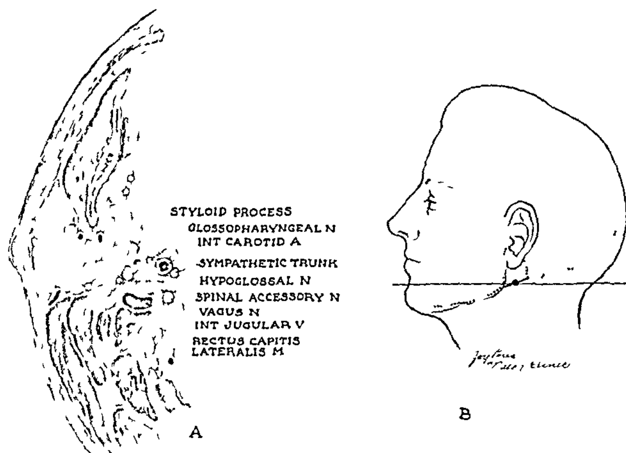


Figure 7 (A) Cross section showing anatomy of the glossopharyngeal nerve the hypoglossal nerve the vagus nerve the accessory nerve and the fibers of the cervical portion of the sympathetic nervous system Their close proximity to each other is responsible for the block of all when an attempt to block any single one is made (B) Level of cross section

other regional blocks of the neck e g superficial or deep cervical blocks [4, 8] Hartel and Keppler [3] and Shaw [4] found that a large number of patients (15 of 17 and 12 of 15 respectively) who had brachial plexus blocks developed a paralysis of the phrenic nerve which could be demonstrated by roentgen studies This is of little significance provided the block is unilateral and the parenchyma of the lungs and/or the muscles of respiration are not markedly affected by other diseases However if a bilateral block is performed in an emphysematous patient and both phrenic nerves are paralyzed by overflow of the solution, acute hypoxia (alveolar and/or tidal) may develop One such case has been seen at the Mason Clinic in which the patient needed continuous oxygen administration by bag and mask in surgery and by tent on return to his room until the block dissipated itself (see Figure 6 page 43) This type of oxygen want would be classified as a tidal hypoxia.

Bilateral Paralysis of the Vagus or Recurrent Laryngeal Nerves—Block of the vagus or recurrent laryngeal nerves may occur during bilateral nerve blocks of the neck particularly superficial deep cervical blocks and stellate ganglion blocks If these nerves are blocked unilaterally usually no respiratory difficulty other than hoarseness occurs However if blocked bilaterally acute oxygen want from adduction of the vocal cords may develop necessitating intubation for the duration of the pharmacological action of the drug This is a tidal hypoxia

Bilateral Paralysis of the Glossopharyngeal Nerve Vagus Nerve the Hypoglossal Nerve

PROPHYLAXIS AND TREATMENT

Following an attempt to perform a bilateral block of the vagus nerves, the glossopharyngeal nerves or the hypoglossal nerves individually at or immediately distal to the foramen ovale, paralysis of all three nerves usually ensues regardless of the care with which block of one of these nerves is performed. The proximity of these nerves to each other usually precludes prophylactic steps to avoid this overflow (see Figure 7, page 41). Treatment of the oxygen want which results from bilateral block of all three of these nerves requires the insertion of an oral airway or an endotracheal tube to avoid hypoxia. The prophylaxis and treatment of oxygen want from the other stated causes vary with the cause and are considered elsewhere in this book (see Chapters 1 and 2, pages 19 and 34, Chapter 3, page 39, and Chapter 6, page 62).

COMMENT

Asthmatic Attacks Following Block of the Cervicothoracic Portion of the Sympathetic Nervous System.—Cases of asthmatic attacks following block of the cervicothoracic portion of the sympathetic nervous system (stellate ganglion block) have appeared in the literature and such attacks would probably cause primary oxygen want.[8]

Etiology.—The exact action of the autonomic nervous system on the bronchi and bronchioles has not been definitely established and it is possible that blocking the sympathetic system allows the parasympathetic fibers to become overactive, thus precipitating an asthmatic attack.[6] On the other hand, stellate ganglion block has been effectively employed to stop status asthmaticus.[7] Therefore, it must be concluded that the etiology is unknown.

In some cases that develop this complication, the history may reveal a hereditary allergic tendency. A true allergy to the blocking agent itself and the consequent liberation of histamine may also be responsible for such an asthmatic attack.

Signs and Symptoms.—Orthopnea, cyanosis, expiratory wheezing and hyperpnea occur. The veins of the neck and face may become engorged.

Treatment.—In addition to oxygen by bag and mask, ¼ cc. of a 1:1000 Adrenalin solution diluted in 5 cc. of normal saline and *slowly* given intravenously or ½ cc of a 1:1000 Adrenalin solution given intramuscularly may stop the attack. If Adrenalin does not halt the attack, Aminophylline 7½ gr. (450 mgm.) to 15 gr. (900 mg.) given *slowly* intravenously usually will do so. Rapid intravenous administration of Aminophylline (theophylline with ethylenediamine) may lead to cardiovascular collapse. These amounts of Aminophylline may be placed in 1,000 cc. of a 5% dextrose in distilled water solution and given by the intravenous drip method. Should the attack be caused by a true systemic allergic reaction to the local anesthetic drug, then Benadryl may be of marked value (see Chapter 2, page 35 for specific dosage).

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anemia In the first situation the patient may appear pale because vasoconstriction produced by the vasoconstrictor drug prevents the reduced hemoglobin from reaching the skin capillaries In the latter instance i e anemia there may not be enough circulating hemoglobin Let us stress that 5 gms of reduced hemoglobin per 100 cc of blood must be present before cyanosis is apparent If a patient has an advanced anemia or has hemorrhaged severely and as a result has only 5 gm of hemoglobin per 100 cc of blood cyanosis cannot appear

Cardiac Failure and Death —Should the physician not recognize the complication blood pressure will fall the pulse will become imperceptible and shock and cardiac failure may ensue Severe hypotension is a late and poor prognostic sign of primary oxygen want and it usually occurs only if the complication goes unrecognized too long or is inadequately treated

TABLE XI

COMPARISON OF OXYGEN WANT IN REGIONAL BLOCK

	PRIMARY OXYGEN WANT	SECONDARY OXYGEN WANT
TYPE OF BLOCK RESPONSIBLE	*Local infiltration* *Peripheral nerve block*	*Spinal block* *Epidural block*
ETIOLOGY	I Obstruction of the respiratory tract (tidal hypoxia) —It results from paralysis of nerves to pharynx larynx and tongue II Nonfunctioning alveoli (tidal hypoxia) —It results from pneumothorax and emphysema III Depression of respiratory center (tidal hypoxia) —It results from systemic reaction to local anesthetic drug IV Increased metabolism (demand hypoxia) —It results from systemic reaction to vasoconstrictor drug	I Hypotension (stagnant hypoxia) —This is the principal cause initially and results from dilatation of peripheral vascular system II Paralysis of muscles of respiration (tidal hypoxia) —As the level of anesthesia rises paralysis of intercostal and phrenic nerves increases oxygen want
SIGNS and SYMPTOMS		
Cortical Manifestations	Same	Same
Respiratory Manifestations	Same	Same
*Cardiovascular Manifestations**		
Blood Pressure	Normal or elevated	Low
Pulse	Initially rapid Later slow full bounding	Initially rapid Later very rapid almost imperceptible weak
Appearance	Cyanotic†	Cadaveric

* As cardiac failure and death approach, blood pressure and pulse disappear in both types of oxygen want

† For cyanosis to appear at least 5 gm of reduced hemoglobin must be present per 100 cc of blood Two conditions in which primary oxygen want can exist without cyanosis are (1) increased metabolism from vasoconstrictor drug (blood does not reach the skin because of the peripheral vasoconstrictor effect of the *vasoconstrictor drug*) and (2) anemia (insufficient hemoglobin to have 5 gms of reduced hemoglobin per 100 cc of blood)

PROPHYLAXIS AND TREATMENT

Following an attempt to perform a bilateral block of the vagus nerves, the glossopharyngeal nerves or the hypoglossal nerves individually at or immediately distal to the foramen ovale, paralysis of all three nerves usually ensues regardless of the care with which block of one of these nerves is performed. The proximity of these nerves to each other usually precludes prophylactic steps to avoid this overflow (see Figure 7, page 44). Treatment of the oxygen want which results from bilateral block of all three of these nerves requires the insertion of an oral airway or an endotracheal tube to avoid hypoxia. The prophylaxis and treatment of oxygen want from the other stated causes vary with the cause and are considered elsewhere in this book (see Chapters 1 and 2, pages 19 and 34, Chapter 3, page 39, and Chapter 6, page 62).

COMMENT

Asthmatic Attacks Following Block of the Cervicothoracic Portion of the Sympathetic Nervous System.—Cases of asthmatic attacks following block of the cervicothoracic portion of the sympathetic nervous system (stellate ganglion block) have appeared in the literature and such attacks would probably cause primary oxygen want.[5]

Etiology.—The exact action of the autonomic nervous system on the bronchi and bronchioles has not been definitely established and it is possible that blocking the sympathetic system allows the parasympathetic fibers to become overactive, thus precipitating an asthmatic attack.[6] On the other hand, stellate ganglion block has been effectively employed to stop status asthmaticus.[7] Therefore it must be concluded that the etiology is unknown.

In some cases that develop this complication the history may reveal a hereditary allergic tendency. A true allergy to the blocking agent itself and the consequent liberation of histamine may also be responsible for such an asthmatic attack.

Signs and Symptoms.—Orthopnea, cyanosis, expiratory wheezing, and hyperpnea occur. The veins of the neck and face may become engorged.

Treatment.—In addition to oxygen by bag and mask, ¼ cc. of a 1:1000 Adrenalin solution diluted in 5 cc. of normal saline and *slowly* given intravenously or ½ cc. of a 1:1000 Adrenalin solution given intramuscularly may stop the attack. If Adrenalin does not halt the attack, Aminophylline 7½ gr. (450 mgm.) to 15 gr. (900 mg.) given *slowly* intravenously usually will do so. Rapid intravenous administration of Aminophylline (theophylline with ethylenediamine) may lead to cardiovascular collapse. These amounts of Aminophylline may be placed in 1,000 cc. of a 5% dextrose in distilled water solution and given by the intravenous drip method. Should the attack be caused by a true systemic allergic reaction to the local anesthetic drug, then Benadryl may be of marked value (see Chapter 2, page 35 for specific dosage).

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CHAPTER 5

High and/or Total Spinal (Subarachnoid) Block Following Peripheral Nerve Block

HIGH or total spinal anesthesia may result as a sequel of the following peripheral nerve blocks (1) paravertebral sympathetic or somatic nerves in the cervical thoracic, lumbar or sacral area (2) the brachial plexus (3) the gasserian (semilunar) ganglion and (4) branches of the trigeminal nerve In most of these instances a dosage of the local anesthetic agent is injected into the subarachnoid space which any physician would agree is massive i e too great a volume and concentration The seriousness of this complication lies in the fact that it occurs when least expected and may go unrecognized While this *complication does not occur frequently it is* by no means rare

ETIOLOGY

The incidence of the high or total spinal *from an inadvertent or accidental spinal in*jection while performing a peripheral nerve block is difficult to ascertain from the reports in the literature which with a few exceptions mention the problem and warn of its possibility but go no further (see Table XII page 49)

The reason for a high or total spinal following a gasserian ganglion block is no mystery the dura or the capsule of the gasserian ganglion has been punctured On the other hand following a peripheral nerve block while the block itself is obviously the initial cause of this complication the question as to how the drug gained entrance to the subarachnoid space is not always easily answered i e did the drug get inside the dura via an incorrectly directed needle a long cuff of dura or the perineural spaces?

Incorrectly Directed Needle—The possibility of the direct placement of a local anesthetic solution inside the dura by an inadvertent *lateral spinal puncture* while performing a paravertebral block must always be kept in mind Although certain anatomical abnormalities may facilitate such placement it is usually caused by too acute a lateral slant of the needle so that the needle passes between the neural arches or through an intervertebral foramen and punctures the dura (see Figure 8 page 50)

Extension of Dural Cuffs Beyond the Intervertebral Foramina—In most instances, the dura and arachnoid fuse with the epineurium of the nerve at or proximal to the intervertebral foramina However on occasion the dura may extend past the foramen before this fusion takes place[3, 6, 8] During myelography we have occasionally noted that the Pantopaque may rapidly pass down what appears to be a long cuff of dura, particularly in the lumbar and sacral areas (see Figure 9 page 50) If such a lateral prolongation of the dura accompanies a nerve being blocked paravertebrally and particularly if paresthesias are sought a subdural injection is possible Bonica told the author of a case in which he was performing a brachial block using the supraclavicular approach after eliciting paresthesias and injecting some of the anesthetic solution spinal fluid was aspirated Brittingham Berlin and Wolff[8] state that "An almost unavoidable hazard of paravertebral nerve

block is an inadvertent injection of the agent used into the subarachnoid space. This probably occurs because of an outward prolongation of the subarachnoid space or in other instances because of movement of the patient and tearing of the tissue after the needle is in place."

Spread to the Central Nervous System and the Spinal Fluid via the Perineural Space Following Intraneural Paravertebral Injections.—A review of the anatomy of the spinal cord, particularly its coverings and their relationship to the coverings of peripheral nerves, shows that the perineural spaces of the peripheral nerves distal to the intervertebral foramina are continuous with the perineural spaces of the nerve roots proximal to the intervertebral foramina. Therefore they are potential spaces by which fluids injected into them may spread central to the spinal cord and the spinal fluid (see Figure 10, page 51).[9, 10]

TABLE XII

REPORTED INCIDENCE OF HIGH OR TOTAL SPINAL ANESTHESIA FOLLOWING INADVERTENT INJECTION OF LOCAL ANESTHETIC AGENTS SUBARACHNOIDALLY

Block	*Author*	*Percentage*
Stellate Ganglion Block	Orkin, Papper and Rovenstine[1]	4 in 186 blocks = 2.2%
	Moore*	0 in 2,000 blocks (Recently 2 epidural blocks ensued.)
	Bonica[2]	1 in 2,300 blocks = 0.04%
Paravertebral Somatic or Sympathetic Block	Orkin, Papper and Rovenstine[1]	0 in 172 blocks (1 case spinal cord was traumatized by needle; no solution was injected, see page 215.)
	Mandl[3]	0 in 2,000 blocks (In 5 cases spinal fluid was obtained; no injection was made.)
	Moore*	0 in 377 blocks
	White, Smithwick and Simeone[4]	3 (Number of cases was not stated.)
	Bonica[2]	3 in 650 blocks = 0.46% (Spinal fluid was observed in 11 cases = 1.8%. Needle was readjusted before injecting drug.)
Brachial Block	Moore*	0 in 1,082 blocks
	Bonica[2]	2 in 3,100 blocks = 0.06%
Cervical Block	Moore*	0 in 704 blocks
	Bonica[2]	1 in 350 blocks = 0.3% (Death occurred within 5 minutes.)
Cranial Nerve Block	Moore*	0 in 186 blocks
	Bonica[5]	Cites 3 cases; gives no percentage
Splanchnic (Celiac Plexus) Block	Moore*	0 in 1,535 blocks (In 6 cases spinal fluid was obtained. Needle was readjusted before injecting drug.)

* Author's series at Mason Clinic

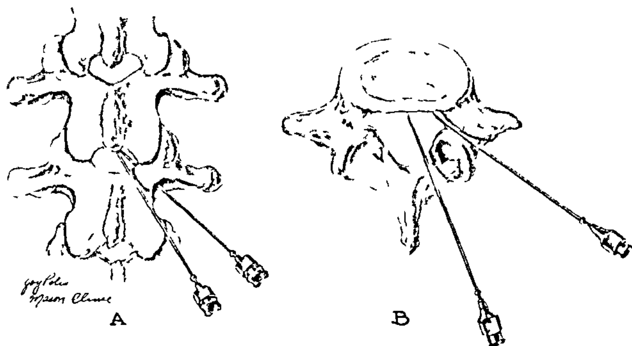


Figure 8 (A) Posterior view of needle entering subarachnoid space either via the space between neural arches or via the intervertebral foramen (B) Cross sectional view of same



Figure 9 Pantopaque in a long cuff of dura covering one of the nerves of the cauda equina This movement of the Pantopaque occurred during myelography when the x ray table was tilted into the vertical position about 10 minutes after the subarachnoid injection of 3 cc of the radiopaque material

Research by Brierly and Field[11] and Key and Retzius[1] using radioactive phosphorus and India ink respectively showed that solutions injected into the peripheral nerves in the leg of an animal can later be detected in the spinal fluid and the parenchyma of the central nervous system Other authors confirm this type of centrad spread[13, 1] Moore *et al*[16] using solutions colored with methylene blue showed that when the lumbar and cervical nerves of monkeys were injected intraneurally 3 to 4 cm from the intervertebral foramen under direct vision the solution spread centrad immediately reaching the parenchyma of the spinal cord in 2 to 5 minutes (see Figure 11 page 52) We further observed that the spinal fluid did not immediately become tinged with the methylene blue It required 10 to 15 minutes for the solution to pass through the pia mater and/or epineurium to stain the spinal fluid lightly but the spinal fluid did not become heavily stained for 35 to 40 minutes Perhaps this latter observation throws light on the reason why the onset of a high spinal may be delayed following intraneural paravertebral injections

Our studies we now believe, indicate that in a number of these cases the physician's placement of the needle was too accurate that is its point rested intraneurally, and that the solution actually spread centrad in the perineural spaces resulting in a high spinal.[16] A case of Adriani[6] may represent such an injection

"Case 2.—A 35 year old white man had received stellate ganglion blocks three days in succession from a senior house officer. Procaine (1 percent) was injected by the anterior route for an injury of the upper arm involving the brachial artery. On the fourth day the block was assigned to a junior member of the house staff. After injecting 10 cc of procaine the operator left the patient to attend another patient. Several minutes later he was called back by the nurse because the patient was not "doing well." The patient was cyanotic pulseless and not breathing. Seven and one half grains of Sodium Amytal were administered intravenously on the premise that a procaine reaction had occurred. The patient did not respond. Manual artificial respiration was instituted by alternately compressing the thorax until a mechanical resuscitator was obtained. All attempts at resuscitation were futile. The operator recalled that while the procaine was being injected the patient complained of a headache. This is a significant observation which should have cast suspicion on the possibility of an intrathecal injection. Valuable time was wasted in administering Sodium Amytal before instituting artificial respiration. Furthermore the Sodium Amytal was not indicated in the absence of convulsive manifestations or excitement."

It is of interest to note that in discussing this case Adriani *et al*[6] stated "The fact that no spinal fluid is withdrawn on aspiration is

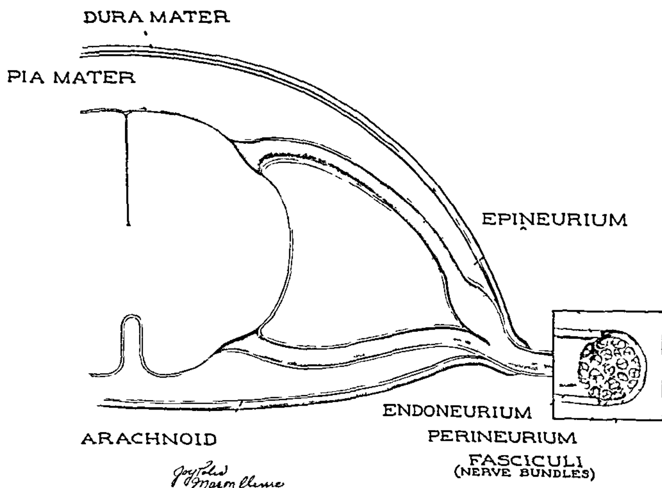


Figure 10 Cross section of the spinal cord. Note that the epineurium of the nerve is a continuation of the pia mater. This relationship essentially provides a pathway for fluid injected intraneurally to undergo centrad diffusion to reach the spinal cord. The interstices between nerve bundles (fasciculi) are filled with connective tissue called perineurium which are potential avenues for retrograde diffusion of materials injected intraneurally

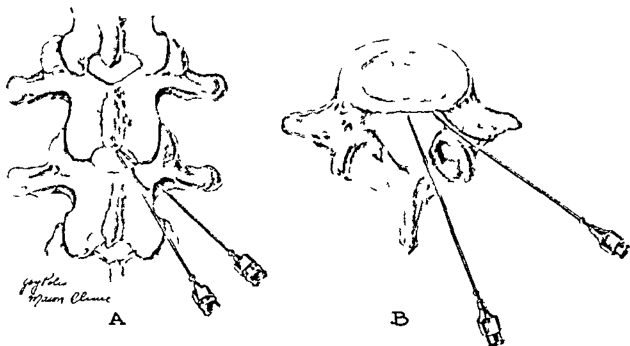


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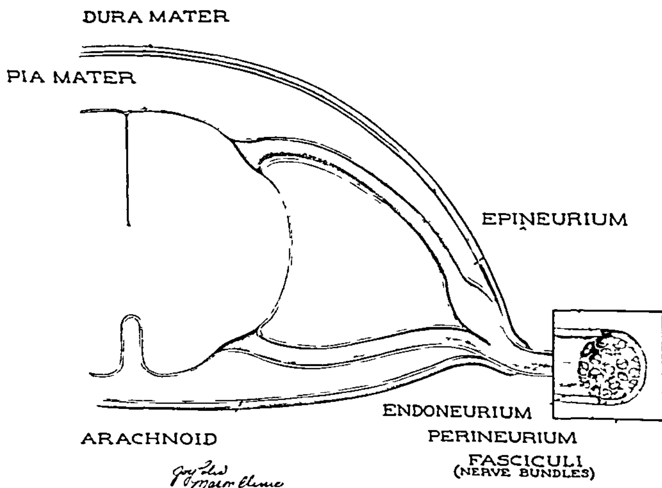


Figure 10. Cross section of the spinal cord. Note that the epineurium of the nerve is a continuation of the pia mater. This relationship essentially provides a pathway for fluid injected intraneurally to undergo centrad diffusion to reach the spinal cord. The interstices between nerve bundles (fasciculi) are filled with connective tissue called perineurium which are potential avenues for retrograde diffusion of materials injected intraneurally.



Figure 11 Dissection of the lumbar area of a monkey showing the centrad spread of an Efocaine methylene blue solution into the spinal cord following an intraneural injection of the solution into a peripheral nerve approximately 4 5 cm from the intervertebral foramen The position of the needle in the nerve cephalad to the injected nerve indicates the point at which 1 5 cc of the colored solution was injected

no assurance that the needle is not in the intrathecal space Not only stellate ganglion blocks sometimes result in total spinals where spinal fluid is not aspirated Similar cases have been reported following block of the brachial plexus the cervical intercostal or the lumbar nerves In all these instances the physicians attributed the complication to the faulty placement of the needle point inside the dura either in the vertebral canal or in a long cuff of dura which extends beyond the intervertebral foramina Could not these injections in retrospect actually be judged perineural injections?

SIGNS AND SYMPTOMS

Generalized severe systemic toxic reactions to local anesthetic drugs characterized by circulatory and respiratory depression without convulsions may be confused with a high or total spinal anesthesia Even the experienced anesthesiologist may frequently find it difficult to differentiate promptly between the two particularly in a case where a large volume of the local anesthetic solution is used Establishment of a definite diagnosis at the onset of such a situation [illegible] essential because the initial therapy [illegible] regardless of its etiology

The onset of a high or total spinal block following an inadvertent, unrecognized spinal varies depending on how the drug entered the subarachnoid space and where the block was performed If the drug is injected directly into the subarachnoid space the onset is usually rapid but if it spreads centrad via the perineural spaces the onset may be delayed In addition a more cephalad injection brings about a more rapid onset For example if an injection of 10 cc of 2 0% Novocain is given inadvertently subarachnoidally while performing a stellate ganglion block a total spinal would rapidly occur But if the same dosage were inadvertently given subarachnoidally during a transsacral block a low or moderately high spinal not a total spinal would in all probability develop depending on the patient s position during and immediately following the block In the latter instance even if a high and/or total spinal anesthesia did develop its onset would in all probability be slower Regardless of the cause of a high or total spinal i e whether it follows an inadvertent puncture of the dura or a purposely administered spinal anesthesia the signs and symptoms are the same (see Chapter 19 page 172)

PROPHYLAXIS

In order to prevent a high or total spinal following a peripheral nerve block the physician must adhere meticulously to proper technique for performing these procedures In addition the following precautions may prevent a high or total spinal anesthesia or warn of the possibility of one developing

Observe the Needle for Drops of Spinal Fluid Appearing at Its Hub—Once the needle is in position for a peripheral nerve block in the vicinity of the vertebral column and before the drug is injected the hub should be carefully watched 15 to 30 seconds for the appearance of spinal fluid

Frequently Aspirate—During paravertebral block frequent attempts to aspirate at least in two planes and preferably four planes should be made while injecting the local anesthetic solutions to be sure the needle has not been advanced into the subarachnoid space

Inject Carefully—During *paravertebral* blocking irrespective of whether or not the patient complains of paresthesias during the placement of the needle or during injection it is wise to move the needle slightly forward and backward so as not to deposit the entire volume of the solution intraneurally This has been termed the "shaking hand technique" *

If the needle is not moved in this fashion then the drug may spread centrad either in the perineural spaces or a prolonged cuff of the dura which extends past the intervertebral foramen This is particularly likely if the patient complains of painful paresthesias as the solution is discharged from the syringe

Stop Injecting if Headache Occurs—If the needle has impaled a cervical nerve during paravertebral blocking in the cervical region headache may occur as the solution is injected Here again if the injection is continued a high spinal may result (see Adriani s case page 51)

Inject a Test Dose—When performing paravertebral nerve blocks it is always wise to inject a test dose of several cubic centimeters of the solution of the drug wait a few minutes and then test for unusual or extensive areas of anesthesia before injecting the balance of the contemplated dose If an extensive area of analgesia occurs during diagnostic and therapeutic blocks the procedure should be postponed until another day If such occurs during a regional block procedure for a surgical operation and the area of anesthesia is not adequate for surgery, a small additional amount of the solution may be given in an attempt to obtain the necessary height Or, to be more conservative and safe the block should be discontinued and supplemented with a general anesthetic

Carefully Observe Every Patient—Following all regional blocks the patient must be *carefully* and *constantly* observed for at least one hour If this is not done and the block performed in a nonchalant manner, a high or total spinal anesthesia is more prone to go unnoticed and result in death

TREATMENT

If a high or total spinal anesthesia follows a peripheral nerve block it is usually an unexpected occurrence and since it occurs when least expected it may result in brain damage and/or death Even though a peripheral nerve block is the cause of this complication the treatment is the same as for a high or total spinal which has followed a purposely administered spinal block (see Chapter 19 page 174)

COMMENT

Inadvertent High Epidural Block—When performing paravertebral somatic or sympathetic blocks in the cervical thoracic or lumbar regions a misplaced needle may result in epidural anesthesia This has occurred recently in two cases at the Mason Clinic following a stellate ganglion block and Adriani *et al*[6] also report a similar occurrence These three patients complained mainly of the inability to use their arms There was no

* Term coined by John S Lundy M D and Albert Faulconer M D Mayo Clinic Rochester Minnesota



Figure 11. Dissection of the lumbar area of a monkey showing the centrad spread of an Efocaine methylene blue solution into the spinal cord following an intraneural injection of the solution into a peripheral nerve approximately 4.5 cm. from the intervertebral foramen. The position of the needle in the nerve cephalad to the injected nerve indicates the point at which 1.5 cc. of the colored solution was injected.

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The onset of a high or total spinal block following an inadvertent, unrecognized spinal varies depending on how the drug entered the subarachnoid space and where the block was performed. If the drug is injected directly into the subarachnoid space, the onset is usually *rapid, but if it spreads centrad via* the perineural spaces, the onset may be delayed. In addition, a more cephalad injection brings about a more rapid onset. For example, if an injection of 10 cc. of 2.0% Novocain is given inadvertently subarachnoidally while performing a stellate ganglion block, a total spinal would rapidly occur. But if the same dosage were inadvertently given subarachnoidally during a transsacral block, a low or moderately high spinal, not a total spinal, would *in all probability* develop, depending on the patient's position during and immediately following the block. In the latter instance, even if a high and/or total spinal anesthesia did develop, *its onset would in all probability* be slower. Regardless of the cause of a high or total spinal, i.e., whether it follows an inadvertent puncture of the dura or a purposely administered spinal anesthesia, the signs and symptoms are the same (see Chapter 19, page 172).

Pneumothorax

PNEUMOTHORAX may occur following any procedure in which a needle is placed in the vicinity of the thoracic cage, i.e., block of the brachial plexus, the stellate ganglion, the intercostal, cervical or phrenic nerves, and of the paravertebral somatic or sympathetic nerves. The frequency of this complication varies greatly depending on the type of block being performed, the approach being used and the skill of the physician executing the block. While numerous articles and books describe and advocate blocks even though they are followed by this complication, only a few give factual material concerning the incidence of pneumothorax (see Table XIII, page 56).[1-6] This is unfortunate because "cloak room" discussion by those who have not used a specific regional technique enough times to become adept often creates an erroneous impression of the incidence of pneumothorax. It is the opinion and experience of the author and Bonica[21] that most regional block procedures in the region of the thoracic cage do not result in pneumothorax often enough to preclude their *everyday routine use in surgery, therapy and diagnosis*. The exception to this is paravertebral thoracic sympathetic block which, even in the hands of the expert, is a singularly difficult block to perform without producing a pneumothorax since the lung and pleura lie in close contact with the bodies of the thoracic vertebrae (see Figure 12, page 55).

ETIOLOGY

Pneumothorax is the presence of air in the pleural cavity. During or following a regional block procedure air may enter the pleural cavity in two ways: either through the needle as it pierces the parietal pleura or via a rent in the lung caused by the needle point puncturing the lung tissue.

Piercing of the Parietal Pleura.—The entrance of air into the pleural cavity via the needle is always a possibility, although a highly unlikely one because (1) it has proven a difficult maneuver in tuberculosis sanitoria when therapeutic pneumothoraces are purposely performed; (2) although needle bi-

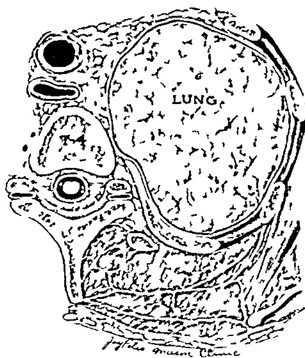


Figure 12. Cross section at the level of T_4 showing the relationship of the pleura to the body of the vertebra, the transverse process and the ribs. This clearly illustrates why a pneumothorax can easily result during an attempt to block a thoracic sympathetic ganglion.

fall in blood pressure or any other untoward result In our cases 10 cc of 0 25% Pontocaine with 0 1 cc of Adrenalin 1 1000 had been used and the patient s arms were paralyzed for approximately three hours

Usually when an epidural block results from the above mentioned blocks the conse quences are not serious because the volume of solution injected is seldom large enough to produce an extensive epidural block There fore treatment other than watchful waiting for the signs and symptoms to clear is not necessary If hypotension and oxygen want develop they must be treated just as if a high spinal anesthesia had developed (see Chapter 19 page 174)

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CHAPTER 6

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Pneumothorax is the presence of air in the pleural cavity. During or following a regional block procedure air may enter the pleural cavity *in two ways*: *either through the needle* as it pierces the parietal pleura or via a rent in the lung caused by the needle point puncturing the lung tissue.

Piercing of the Parietal Pleura.—The entrance of air into the pleural cavity via the needle is always a possibility, although a highly unlikely one because (1) it has proven a difficult maneuver in tuberculosis sanitoria when therapeutic pneumothoraces are purposely performed; (2) although needle bi-

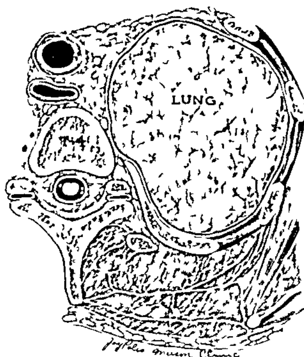


Figure 12. Cross section at the level of T_4 showing the relationship of the pleura to the body of the vertebra, the transverse process and the ribs. This clearly illustrates why a pneumothorax can easily result during an attempt to block a thoracic sympathetic ganglion.

fall in blood pressure or any other untoward result In our cases 10 cc of 0 25% Pontocaine with 0 1 cc of Adrenalin 1 1000 had been used and the patient's arms were paralyzed for approximately three hours

Usually when an epidural block results from the above mentioned blocks the consequences are not serious because the volume of solution injected is seldom large enough to produce an extensive epidural block Therefore treatment other than watchful waiting for the signs and symptoms to clear is not necessary If hypotension and oxygen want develop they must be treated just as if a high spinal anesthesia had developed (see Chapter 19 page 174)

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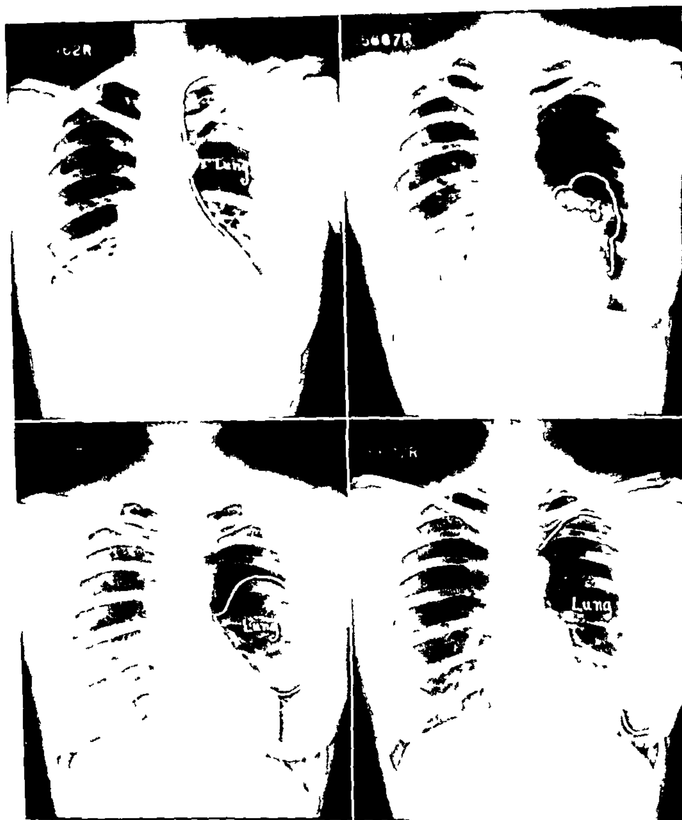


Figure 13 This patient complained of pain in her chest at the time of the brachial block (A) (*Top left*) X ray taken 6 hours after the block had been performed and the surgery completed shows no evidence of pneumothorax The difference in density of the left chest in this x ray is caused by the shadow of the scapula not air in the chest (B) (*Top right*) X ray taken the following day shows an almost complete pneumothorax Following this film 1000 cc of air was withdrawn from the chest (C) (*Bottom left*) X ray taken 4 days following film B after repeated withdrawal of 1000 cc of air from the chest on successive days (D) (*Bottom right*) X ray taken 2 days after film C shows lung 80% expanded

opsies of the lung with 18 gauge needles have often been performed, the incidence of pneumothorax in these cases is extremely small [5, 53] (3) pneumothoraces following regional block procedures may not occur for two to six hours after the completion of the block (see Figure 13, page 57), [34, 37] and (4) most physicians, when performing regional procedures where pneumothorax is possible do not use a needle with an open hub—a syringe is attached a

TABLE XIII

REPORTED INCIDENCE OF PNEUMOTHORAX FOLLOWING REGIONAL BLOCK IN ARTICLES REVIEWED[1-60]

Block	*Author*	*Percentage of Pneumothorax*
Brachial Plexus	Griswold & Woodson[29]	2 in 100 blocks = 2%
	Dmarjian[30]	1 in 100 blocks = 1%
	Bonica Moore & Orlov[31]	9 in 1000 blocks = 0 9%
	Bonica & Moore[32]	10 in 1 512 blocks = 0 66%
	Macintosh & Mushin[33]	3 in 400 blocks = 0 75%
	Moore & Bridenbaugh[34]	1 in 100 blocks = 1 0%
	Small[35]	0 in 151 blocks
Stellate Ganglion	Moore*	1 Anterior (paratracheal) approach *0 in 2 000 blocks*
		2 Anterolateral approach 2 in 25 blocks = 8%
	Orkin Papper & Rovenstine[37]	1 Anterior (paratracheal) approach 0 in 27 blocks
		2 Anterolateral approach 3 in 151 blocks = 1 9%
		3 Posterior approach 1 in 8 blocks = 13%
	Bonica[51]	1 Anterior (paratracheal) approach 5 in 2 000 blocks = 0 25% (All 5 occurred with Arnulf's approach)
		2 Anterolateral approach 1 in 200 blocks = 0 5%
		3 Posterior approach 3 in 100 blocks = 3%
Intercostal	Chivers[38]	19% (estimated)
	Moore*	1 in over 2 188 blocks = 0 05%
	Bonica[51]	0 in 750 blocks
Paravertebral thoracic somatic	Moore*	0 in 50 blocks
	Bonica[51]	17 in 370 blocks = 4 6%
Paravertebral sympathetic thoracic	Orkin Papper & Rovenstine[37]	4 in 172 blocks = 2 33%
	Moore*	1 in 20 blocks = 5 0%
	Mandl[46]	5 in 350 blocks = 1 42%
	Kappis & Gerlach[43]	4 in 100 blocks = 4%
	White[50]	3 in 100 blocks = 3%
	Bonica[51]	22 in 280 blocks = 7 85%
Cervical	Moore*	0 in 704 blocks
	Bonica[51]	0 in 350 blocks
Phrenic	Moore*	0 in 35 blocks
	Bonica[51]	0 in 63 blocks

* Author's series at Mason Clinic

tation and percussion may not reveal the cause (see Figure 13 page 57) Examination should be repeated in 6 hours if pain persists Some patients have not complained of pain until 12 hours following the block The pain is usually sharp and therefore, decreases the depth of respirations

Anxiety.—When pain develops most patients show anxiety and this may contribute to the signs of oxygen want by increasing oxygen demand

Oxygen Want.—Dyspnea may or may not occur depending on the degree of collapse In our cases of unilateral pneumothorax most of the patients had dyspnea initially but this usually subsided unless the patient had some complicating disease such as anemia emphysema etc *Orthopnea* and *cyanosis* (5 gm of reduced hemoglobin per 100 cc of blood) seldom result

On the other hand, if a bilateral pneumothorax develops dyspnea orthopnea and cyanosis are likely occurrences

Hypotension.—There may or may not be a change in the blood pressure indicating respiratory embarrassment or shift of the mediastinum Should a complete *unilateral pneumothorax* result during a block procedure a temporary hypotension may ensue initially This is presumably due to a shift of the mediastinum—not to the oxygen want alone and in the *normal healthy adult* a compensatory reflex usually corrects the hypotension In these cases whether or not hypotension is found depends to a great extent on the rapidity with which the pneumothorax develops

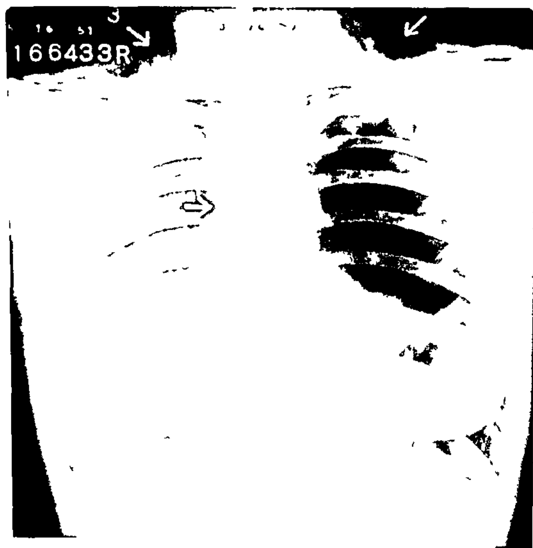


Figure 14 Pneumothorax with mediastinal and subcutaneous emphysema following a brachial block Arrows mark areas of emphysema

stylet is in place or a finger is held over the opening in the hub until the needle is correctly placed

Piercing of the Lung Tissue —Macintosh and Mushin [6] Bonica [4] Wishart [49] and the author [34, 4] feel that *most pneumothoraces* are caused by the piercing of the lung substance proper by a misplaced needle If this misplacement is not immediately rectified inhalation and exhalation move the lung surface and it is torn by the needle point Either the initial hole or the rent in the lung allows subsequent air leakage

While it is evident that the puncture of the pleura or lung tissue must take place for a pneumothorax to result from a regional block procedure enlargement of the needle puncture exertion in the immediate postblock period etc *are often predisposing factors*

Predisposing Factors —The following have been mentioned as predisposing factors and must be given careful attention when performing blocks in the region of the thoracic cage

Height the Pleura Extends into Neck — This is of particular importance during block of the brachial plexus stellate ganglion phrenic nerves and cervical nerves In general the pleura extends higher into the neck in the tall thin person than in the average or short fat person [34] Also the right cupola of *the pleura is normally higher by one to two* centimeters than the left

Chronic Emphysema — Wishart [49] points out that special care must be exercised in performing brachial plexus block on patients suffering from chronic emphysema because if a bulla is punctured it does not seal off as rapidly as would the normal lung and a severe tension pneumothorax with marked respiratory and cardiac embarrassment may ensue

Motion of the Patient Straining and Increase in Respiratory Rate or Depth —During the block procedure if the patient is not correctly medicated prior to the block procedure or if the procedure is not carefully explained to him prior to and during its execution he may move suddenly and cause the needle to enter the lung If the patient is excited an increase in the rate or depth of respirations may result and if the needle enters the lung at this time there is a greater chance that a relatively large rent will result than if the respiratory movements are normal or retarded

After the block has been completed and the operation accomplished physical exercise resulting in an increase in the respiratory rate and depth may cause a pneumothorax when the lung has inadvertently been punctured On the contrary if a patient whose lung has been pierced inadvertently is placed at full bed rest and sedated to decrease the respiratory rate and depth a pneumothorax is less likely to develop (see page 63)

SIGNS AND SYMPTOMS

If the following occur while performing a block in the region of the thoracic cage one may assume that the lung has been punctured and a pneumothorax might result (1) the needle inadvertently slips off the rib or is placed too deep in an attempt to find bony landmarks (2) a sucking noise is heard (3) air is aspirated (4) the patient coughs or complains of chest pain and (5) the solution injected is tasted by the patient All of these have happened at the Mason Clinic yet only in a very few of these patients have the signs and symptoms of a pneumothorax developed *This definitely proves that the lung may be* punctured without a pneumothorax of significant degree resulting However the physician should not draw a sigh of relief if a pneumothorax does not develop immediately in these patients Some of our patients have not developed the signs and symptoms of this complication until 6 to 12 hours after the completion of the block (see Figure 13 page 57)

The severity of a pneumothorax usually but not always depends upon the degree of collapse and one or more of the following signs and symptoms may result

Pain —The first indication is pain in the chest accentuated by deep breathing This may be the only sign that the pleura and *lung have been invaded Initial x ray auscul*

tation and percussion may not reveal the cause (see Figure 13 page 57) Examination should be repeated in 6 hours if pain persists Some patients have not complained of pain until 12 hours following the block The pain is usually sharp and therefore decreases the depth of respirations

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Figure 14 Pneumothorax with mediastinal and subcutaneous emphysema following a brachial block Arrows mark areas of emphysema

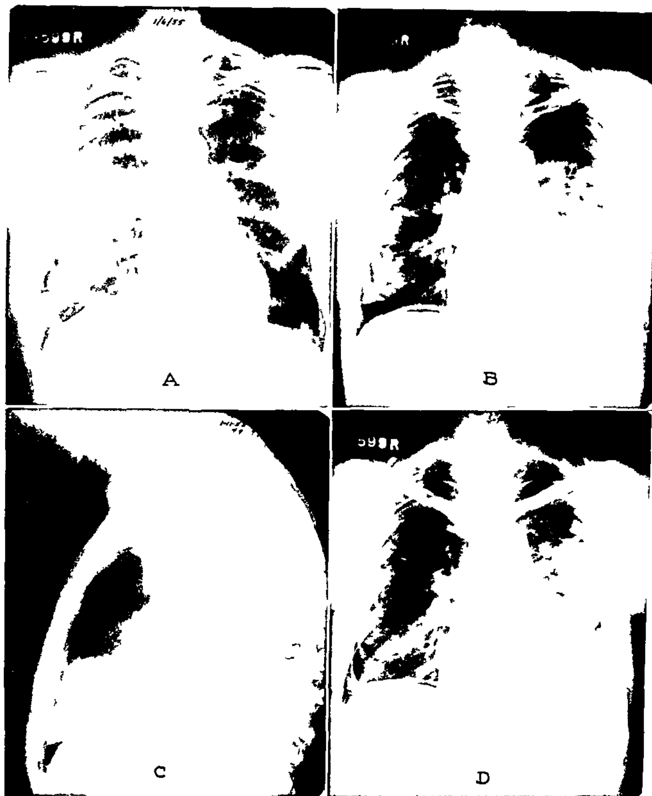


Figure 15 Pleural effusion and a pneumothorax (20% or less) following intercostal splanchnic (celiac plexus) nerve block for an enbloc resection of the transverse colon gastrojejunostomy site the stomach and the spleen for an adenocarcinoma of the stomach arising at an old gastrojejunostomy site in a 60 year old male (A) Preoperative chest film (B) Posterior anterior view of the pneumothorax and pleural effusion Dotted line indicates upper border of lung (C) Lateral view of pneumothorax and pleural effusion (D) Posterior anterior view of chest following withdrawal of 500 cc of serous sanguinous fluid and 2000 cc of air X ray department s report of this last film read "The left lung is now completely expanded The left lower lobe is slightly decreased in size Small amount of fluid in the left pleural space still obscures the diaphragm on that side posteriorly Picture is that of general improvement All films are taken at a distance of 6 feet

However in the patient with a poor *physical status*, a hypotension as a result of the oxygen want may follow a unilateral pneumothorax

When a *bilateral pneumothorax* occurs corrective therapy is essential whatever the patient's physical status, and adequate steps to correct the oxygen want are necessary, otherwise hypotension and death may result A persistent hypotension in such a situation is usually a *late* sign of oxygen want

Tachycardia—If oxygen want or hemopneumothorax occurs they may be accompanied by an increased pulse rate[3]

Auscultation, Percussion and Failure of the Chest to Rise on the Blocked Side During Inspiration—Auscultation and percussion usually are of little help in determining whether or not a pneumothorax has occurred unless more than a 20% collapse has resulted Then breath sounds may be absent decreased or distant and percussion may reveal air in the pleural cavity on the affected side

Failure of the chest to rise on the side blocked may or may not be of significance because the diaphragm will not move if the phrenic nerve is anesthetized following a cervical stellate ganglion or brachial block Therefore on inspiration the chest on the blocked side may not rise as much as on the unblocked side although no pneumothorax has resulted This is also true if the intercostal nerves particularly the lower ones (T4 T12) have been anesthetized

Hemoptysis—To date we have not seen

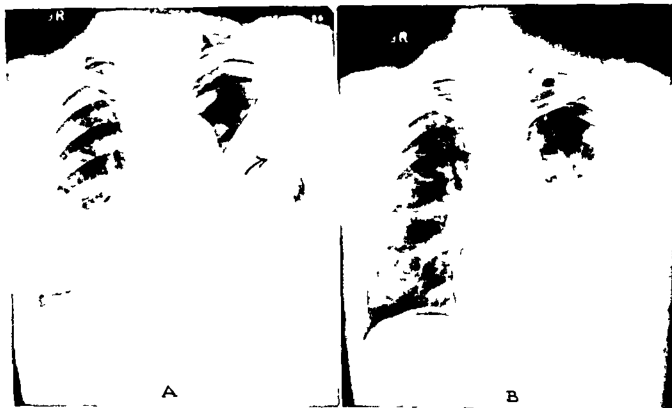


Figure 16 Comparison of film taken at the bedside (A) and one taken at a distance of 6 ft in the x ray department (B) The x ray department's report of the bedside x ray read Comparison with film of 1 6-55 (see Figure 15 A page 60) demonstrates postoperative air under the right diaphragm and a collapse of the left lung with fluid in the lower pleural space The collapse is due to pneumothorax All of these changes have developed since 1 6-55 In addition there might be slight aspiration in the right middle lobe That for the 6-foot film stated 'This film better illustrates the pneumothorax and hydrothorax in the left chest The fluid level lies just under the third rib anteriorly and the expanding upper lobe crosses the dependent portion of the 1st rib anteriorly The impression is gained that there is slightly greater expansion than on the portable film There is air under the right diaphragm Heart is displaced somewhat to the right by fluid and possibly by positive pressure from the pneumothorax Right lung is clear By comparing the 2 films it will be noted that a more certain diagnosis can be made from the 6-foot film Arrow on (A) shows fluid in one of the fissures of the lung The dotted line indicates the upper border of the lung

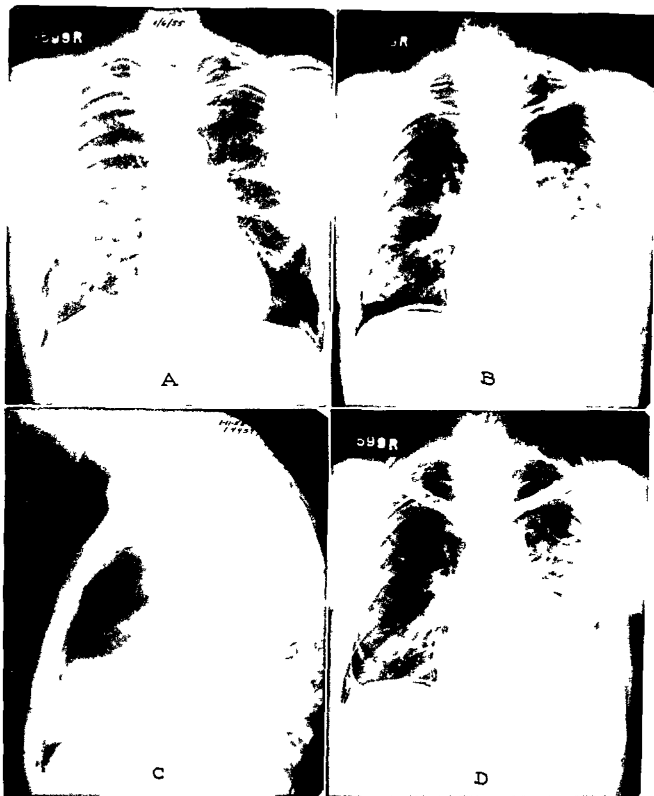


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the pleura out of the way as the needle is being advanced.[4] We have not used this maneuver because it interferes with the anesthetist's tactile sense of what the needle point is striking.

Full Cooperation of the Patient During the Block Procedure.—To assure full cooperation of a patient during a block procedure he should be seen prior to the block so that the procedure may be carefully explained to him and the correct premedication ordered. During the block he should be impressed with the necessity of not jumping, and should be warned beforehand of any painful procedure. This is particularly important when performing a block where paresthesias are essential and light premedication, i.e., morphine sulfate gr 1/6 (10 mg.) and atropine gr 1/150 (0.4 mg.), but no barbiturate is used. Sudden motion by the patient as the needle is being advanced will often result in deep penetration and puncture of the parenchyma of the lung.

It has been our custom in performing intercostal deep splanchnic (celiac) block for upper abdominal surgery to anesthetize the patient lightly with a rapid drip of 0.6% Pentothal (thiopental) to the point where the lid reflex is lost and maintain him in this state until the block is completed. This assures that the patient will not jump during the procedure and decreases the amplitude of respiration. We attribute the low incidence of pneumothorax (1 in 1445 = 0.07 per cent) in these particular cases to this type of sedation.

Rest Needle on Rib When Refilling Syringe.—When refilling the syringe during intercostal nerve block and brachial plexus block the needle point should rest on the rib. This precaution prevents puncture of the lung should the patient move.

Bed Rest Following Blocks.—If it is suspected that the lung has been punctured during a block procedure, i.e., the needle inadvertently slips off rib, the patient coughs during the block, or air is aspirated, etc., the patient should be put at bed rest for 10 to 24 hours and heavily sedated to give the puncture time to seal and stay sealed. Experience has confirmed this recommendation. For example: A patient from one of the outlying small towns came to Seattle to shop. When stepping from the bus she fell and sustained a Colles' fracture at 8:00 A.M. She received a brachial block at 9:00 A.M. During the procedure the needle slipped off the rib and the patient coughed. The block was completed, the fracture set, and the patient returned to the recovery room. She was seen by the Anesthesia Department at 12:00 Noon. There were no signs or symptoms of a pneumothorax, and because she pleaded to be allowed to continue her shopping trip she was discharged. At 8:00 P.M. she called and said she had one hour before developed a severe pain in her chest on the side of her fractured arm. She gave us the story that after leaving the hospital she leisurely shopped, not exerting herself, but she had to run to catch her bus and stand up part of the way home. Shortly after catching her bus the severe pain developed. It seems fairly obvious from this story that the hole in the lung made at the time of the block had immediately sealed itself and remained sealed until excessive physical exertion occurred, accompanied by a marked increase in the depth and rate of respiration. This broke the seal and a pneumothorax resulted. X-ray revealed a 60% collapse. It is our belief that had this patient remained relatively inactive, preferably at total bed rest, and had she been well sedated the pneumothorax would not have occurred. The use of bed rest and sedation can not be too strongly urged in the prophylaxis of pneumothorax.

TREATMENT

The treatment of pneumothorax is entirely symptomatic while the patient is in the operating or block room. Definitive treatment can not be decided upon until the degree of pneumothorax is determined.

Immediate Symptomatic Treatment.—When a pneumothorax occurs the patient usually becomes very apprehensive about his condition and must be soothed, if at all possible, by

thus but one of Wishart's[49] cases exhibited it as the only sign of pneumothorax and Mandl[46] observed it for several days following puncture of the lung

Subcutaneous and Mediastinal Emphysema—At times air may be felt in the tissue at the site of the insertion of the needle and occasionally a marked subcutaneous and mediastinal emphysema may occur (see Figure 14 page 59) Usually this is of no consequence However Capelle[27] reports a case of pneumothorax following brachial block in which a severe emphysema and death occurred

Pleural Effusion—Judging from the literature reviewed pleural effusion following a pneumothorax from a regional block procedure is evidently infrequent At the Mason Clinic we have seen only one such case and this followed an intercostal splanchnic (celiac plexus) block for an enbloc resection for carcinoma of the stomach (see Figure 15 page 60)

In this case the spleen was found to be adherent to the diaphragm and had to be removed intracapsularly It is possible that diaphragmatic irritation caused by this surgery may have been a contributing factor to the resulting effusion Nevertheless the pneumothorax which was caused by the block itself probably initiated the effusion It should also be noted that at the time of the block and during surgery there was no reason to suspect that the needle had entered the lung Therefore at the completion of surgery (3½ hours) the patient underwent a routine bronchoscopy and a reasonable amount of thick mucus was aspirated from the bronchi Being awake at the time of bronchoscopy he coughed vigorously and it is possible that this episode of coughing may have reopened a hole in the lung which had been made previously and had sealed

In this case 500 cc of a serous sanguinous fluid was aspirated along with an estimated 2 000 cc of air and the patient had an uneventful recovery from the pneumothorax (see Figure 15D page 60) The pathologist estimated that the effusion fluid contained 5% hemoglobin In this case the pneumothorax may well have expanded by itself but it is our opinion that effusion is a definite indication for aspiration If the fluid is not withdrawn it may organize form a pocket of fluid or adhesions and necessitate lung surgery in the future

X ray Evidence—Whether or not a patient has a pneumothorax can be established conclusively only by x ray If the patient shows the signs and symptoms of a pneumothorax a six foot x ray should be taken immediately if surgery is cancelled or after surgery is completed to ascertain the extent of the pneumothorax If no pneumothorax is found the x ray should be repeated in 6 to 12 hours (Figure 13 page 57) *Bedside x rays are misleading uncertain cause improper treatment are a waste of the x ray department's time and the patient's money and should not be taken because they often lead to misdiagnosis* (see Figure 16 page 61)

PROPHYLAXIS

Close Adherence to the Techniques—This is perhaps the most important way of avoiding a pneumothorax Since the ribs and vertebrae are used as landmarks in most of the block techniques in the area of the thoracic cage the authors describing such procedures usually *repeatedly emphasize the importance* of keeping the needle point in contact with these structures and stress that it is only when the initial landmark is incorrect or the needle is allowed to wander away from the bone that the lung tissue is invaded[34, 4, 4]

Forceful Expiration When Placing Needles—*Bonica*[47] *advises in reference to paraver*tebral blocks in the area of the thoracic cage that *The patient should also be instructed to* make a forceful expiration and hold it for 10 to 15 seconds so as to remove the lung from the path of the needle while the latter is being advanced toward the lateral surface of the vertebral body

Injection of Saline as Needle Is Being Introduced—*Some have used this technique* occasionally in hopes of exerting sufficient pressure ahead of the needle point to push

lung shows progressive expansion the patient should have a chest x ray at least once a week until complete re expansion has taken place

COMMENTS

Chest Pain Following Block in Region of Thoracic Cage Is Not Always an Indication of Pneumothorax—In a series of 100 consecutive brachial blocks in which x ray studies were made following the blocks and surgery Bridenbaugh and the author[30] found only one pneumothorax although five patients reported pain which resembled the pain of pneumothorax We stated "It is our opinion that in these cases [the four without pneumothorax] the pain was due to stimulation of the long thoracic nerve during the performance of the block or the irritation of the pleura from either excessive spread of the anesthetic solution or trauma from the point of the needle The first mentioned factor seems to be the more likely Pain of this nature invariably disappeared not later than the third postoperative day while it is not uncommon for the pain from a pneumothorax to persist for a week" Mandl[46] substantiates the fact that pleural pain is not infrequent following block in the thoracic region in cases where a pneumothorax cannot be demonstrated

In order to establish the diagnosis of pneumothorax with certainty an x ray must be taken and collapse of the lung demonstrated The author has seen a patient with chronic emphysema exhibit the signs and symptoms of pneumothorax following a bilateral brachial block when in reality the paralysis of both phrenic nerves was responsible (see Figure 6 page 43)

Do Silent Pneumothoraces Occur Following Block Analgesia and if so What Is Their Significance?—The fact as to whether or not "*silent pneumothoraces* occur in any great number following block analgesia seems to us to be of only academic interest since the patient is in no way discommoded It was probably proposed as a possible complication of block analgesia because many patients with spontaneous or therapeutic pneumothoraces are asymptomatic Nevertheless since the term has been mentioned we believe that our studies on this subject reveal the following (1) air in the chest detectable by x ray after brachial block caused pain and was never silent and (2) "silent" pneumothorax following brachial plexus block analgesia is probably hypothetical in the healthy adult[36]

Bilateral Pneumothorax—This is a very serious complication and must be treated rapidly, or death will result For this reason most authors advise against bilateral blocks in the area of the thoracic cage The only death in the authors series of over 19 000 regional block procedures followed a bilateral thoracic sympathetic block Although the autopsy in this case produced no findings it is the authors opinion that death was probably due to a bilateral pneumothorax

Death from Pneumothorax—Deaths from pneumothorax following regional block for surgical procedures or manipulations are rare in the healthy adult Most of the deaths reported from pneumothorax have occurred in patients receiving diagnostic or therapeutic blocks In these instances the disease for which the block was being attempted such as angina pectoris may have contributed directly or indirectly to the fatalities[33,37,47] The exception to this is the case of Capelle[27] cited previously (see page 62)

Hemopneumothorax—This is very unusual and only one report (by Orkin *et al*[37]) of this complication was found In this case following a cervicothoracic sympathetic block for a Volkmann's contracture the patient complained of chest pain immediately following the block but no pneumothorax was detectable by x ray at that time However the next day a fluid level to the eighth thoracic vertebra and a complete collapse of the lung were found by x ray Aspiration of the chest yielded 1500 cc of blood Even though attempts were made to completely *reinflate the* lung the left upper lobe remained collapsed until a decortication was performed The authors explain this complication by stating It was believed that in the collapse of the

assuring him that everything will be all right

Oxygen Want—The patients immediate *dyspnea* may be relieved by the administration of oxygen The oxygen should not be given under pressure because this would reopen or prevent closing of the rent in the lung

Pain should be alleviated with intravenous morphine sulfate gr 1/6 to gr 1/4 (10 to 15 mg) or Demerol 50 to 100 mg or any other opiate of the physicians own choice if the initial discomfort is marked

Hypotension—If hypotension occurs it should be treated as indicated in Chapter 15 page 147

Make Plans for Subsequent Therapy—As soon as the patient has been made comfortable by this symptomatic treatment and the operation for which the block was performed executed or cancelled a six foot x ray should be taken to determine the extent of the collapse If no pneumothorax is present but it is known that the lung was punctured x ray should be repeated in 6 to 12 hours (see Figure 13 page 57) Remember that bedside films are of little value

Treatment of a Pneumothorax of Twenty Per Cent or Less—*Re expansion of the lung* This will take place by itself and catheter or needle drainage is not necessary

Oxygen Want—Once the dyspnea has passed no treatment is necessary

Pain—Codeine gr 1/2 (30 mg) and aspirin gr 10 (600 mg) may be taken as necessary for relief of pain and discomfort Usually this is necessary for only 1 to 3 days although mild pain may persist for 4 or 5 days

Check of Patient—After 10 days have passed an x ray of the chest should be taken to be sure re expansion has occurred To date we have not seen a single case with up to 20% collapse which has not expanded in this period of time Nevertheless while most pneumothoraces complicating regional block clear quickly without sequelae the degree of collapse should be watched closely until expansion of the collapsed lung is complete

Treatment of Pneumothorax of More Than Twenty Per Cent—These cases must not be treated lightly Conscientious and intensive therapy must be instituted to prevent secondary complications such as pneumonia or empyema

Re expansion of Lung—If the collapse exceeds 20% but is not greater than 50% watchful waiting is permissible for 24 to 48 hours If after that time no expansion has taken place needle drainage of the chest is indicated All the cases we have seen that exceeded 20% collapse showed an almost complete collapse of the involved lung (see Figure 13 page 57) and required withdrawal of air from the pleural cavity to start lung expansion Such needle aspiration should be established in the second intercostal space in the midclavicular line Usually only 1000 cc of air are withdrawn at one time so as not to cause rapid shifts of the mediastinum Once the lung has expanded to 50% it is usually unnecessary to withdraw any more air from the pleural cavity as the lung will continue to expand

In the exceptional case which does not respond to intermittent needle withdrawal of the air it may be necessary to insert a small catheter and exert negative pressure with a Stedman pump

Oxygen Want—Dyspnea may require administration of oxygen by mask or tent until it subsides

Pain—In these cases pain may persist for a week but in most instances codeine gr 1/2 (30 mg) and aspirin gr 10 (600 mg) by mouth will furnish relief Other opiates are seldom needed or indicated once the initial severe pain has been controlled

Antibiotic Treatment—As prophylaxis against pneumonia etc 300 000 to 400 000 units of penicillin should be given daily until the lung is at least 50% expanded Should pneumonia develop then the dosage of penicillin should be increased to 600 000 to 800 000 units daily and if the infection does not respond other broader spectrum antibiotics should be employed

Check the Patient—X rays of the chest should be taken at one to three day intervals to be sure expansion is proceeding After the

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lung small veins at the apex between the visceral and parietal pleura were torn resulting in hemorrhage This case also confirms the need to observe patients with pneumothorax carefully until the lung has completely re expanded

Pleural Effusion from Stimulation of the Pleura Alone—Belinkoff reports four cases of pleural effusion following the use of Novest Oil (Monocaine base 0 02 gm benzyl alcohol 0 05 gm Benzocaine 0 3 gm and oil of sweet almond to 1 0 cc) He states the four cases which developed pleural effusions deserve further consideration All were females upon whom cholecystectomy had been performed three under spinal and one under general anesthesia by two different surgeons In all instances there was only a unilateral intercostal block done on the right side which was successful for relief of post operative pain and in all instances the pleural effusion occurred on the same right side In three of the patients the fluid was absorbed without any interference but in the fourth thoracentesis was done on two occasions to remove a total of 1 100 cc of fluid which was clear and from which no organisms could be cultured

"The etiology of this sterile fluid seems to be some irritant stimulating the pleura Probably when the block was being done some of the fluid was inadvertently injected into the pleural space with the resultant effusion The proximity of the pleura to the intercostal nerve is thus stressed and great care must be taken at each injection to make sure that all is deposited in the tissues and none in the pleural space It had been hoped that the agent used would not stimulate the pleura or be an irritant, but apparently in some cases it did Most likely it was the oily solvent that proved to be the irritating factor"

Pleural Shock—The term "pleural shock" has been used loosely and an adequate definition is difficult to find It probably refers to the development of the signs and symptoms of shock following a block in the region of the thoracic cage when the pleura and the lung have been punctured and a true pneumothorax has been produced

Concerning this problem following brachial block Macintosh and Mushin[**] state, " Pleural shock appears in several older writings In investigating these we find that the injection was made with the patient sitting up Possibly this complication is the same as the fainting to which we have already referred " These authors in their monograph had previously noted that fainting during brachial block "occurred only when the injection was given while the patient was sitting up (see page 29)

Cerebral Air Embolism—Adelman [6] reports a case in which this occurred following an attempted stellate ganglion block as a result of misplacing the needle in lung parenchyma *The episode was characterized by* fresh hemoptysis coma and transient hemiplegia which he feels leaves little doubt that the diagnosis of cerebral air embolism was correct The anterolateral approach was employed No specific therapy was instituted and the patient recovered without any residua

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CHAPTER 7

Death and Cardiac Failure

Deaths following regional anesthesia may be caused by respiratory or by cardiovascular failure or by a combination of these two. In many instances it is difficult to determine the primary cause, but in the final analysis the patient may not be pronounced dead until the heart stops beating. Deaths during regional block procedures have been reported following (1) topical application and local infiltration (see Chapter 1, page 7), (2) stellate ganglion blocks,[1-5] (3) thoracic and lumbar paravertebral blocks,[6-9] (4) caudal block,[10] and (5) spinal blocks.[11-14]

Many instances of death following regional block procedures other than those cited above could be added. But even then it is doubtful that a correct estimate of mortality following regional block could be determined because (1) regional block is often confined to poor risk patients for whom a general anesthetic is considered unsafe, (2) a large number of such deaths are not reported in the literature, (3) the incidence varies depending on how the author defines an anesthetic death as well as what the author is trying to prove, and (4) the estimate would vary depending on whether literature on the pioneering of this type of anesthesia is reported.

The importance of this section on deaths and cardiac failure is not to try to establish their incidence, but to warn that they do sometimes follow even the simplest of regional block procedures, and to stress that when the circumstances which caused them are critically reviewed a number (including the one which occurred at the Mason Clinic and is cited in this chapter) might have been prevented if (1) the complication initially responsible had been recognized immediately, (2) equipment and materials for correcting all types of complications from regional blocks had been immediately available and the physician had used them, and (3) when the complication progressed to cardiac arrest or ventricular fibrillation, manual systole had been instituted immediately. Had manual systole been instituted in our case along with other resuscitative measures we did use, resuscitation might have resulted. On the other hand, it must be admitted that even if manual systole is tried, time lost in instituting it, the poor physical status of the patient, or some other reason might preclude successful resuscitation. Nevertheless, this type of rationalization is no excuse for not entering the chest and performing manual systole when cardiac arrest or ventricular fibrillation occurs.

SUDDEN CARDIAC FAILURE
(Cardiac Arrest and Ventricular Fibrillation)

Cardiac failure may occur suddenly or may be heralded by slowly developing hypotension and oxygen want. In the latter instance the astute conscientious anesthesiologist who is carefully watching his patient will diagnose the problem and may have time to correct it before it has progressed to cardiac failure. Fortunately, such warning is available in most regional block procedures, particularly spinal and epidural block. On the other hand, cardiac failure may occur suddenly without warning or be heralded by such very rapidly developing signs and symptoms that regard

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Irrespective of when cardiac arrest or fibrillation develops during a regional block, cardiac failure does occur and the physician must (1) understand the possible causes of this serious terrifying and sometimes catastrophic complication (2) be able to recognize when it occurs, and (3) be able to institute what at the present time is considered to be the most effective treatment

ETIOLOGY

The exact etiology of sudden cardiac failures following regional block usually is difficult to determine Although the following possible or contributing factors other than the block itself or its complications have been proposed as possible causes, the exact etiology of cardiac failure under these conditions remains undetermined at this time In all probability it is not the result of any one single factor but is caused by a combination of the block one of its complications and/or one or more of the factors enumerated below

Reflex Phenomena—It has been shown that stimulation of the nerve endings of the autonomic nervous system may set up a reflex arc which may in turn cause changes in cardiac rate and rhythm This mechanism may be effected either via the parasympathetic or sympathetic nerves or both

Parasympathetic Nervous System i e the Vagovagal Reflex—It is the belief of Reid and his co workers[16, 28, 29] that the vagus nerve is responsible for mediating the reflexes that result in cardiac failure According to them this reflex arc composed of afferent and efferent fibers of the vagus may be established in any one of three ways first over the vagal network to the vagal center and from there by an efferent path to the heart second by an axon reflex in the vagal system by passing from one branch to another without going centrally and third by irradiation of vagal impulses in which the stimulus originated at vagal nerve endings and passes to a ganglion where it transfers to a branch of the sympathetic system and to the heart[16] They emphasize that the degree to which the vagus is stimulated and the area where it is stimulated are all important in determining the outcome of the stimulation that is whether only bradycardia or bronchial spasm results or cardiac failure ensues They also write *No healthy normal heart* can ever stop from vagal stimulation in the sense that serious symptoms will arise One will never see a carotid sinus syndrome in a healthy heart If the heart has been the site of disease in which the specific tissue in the ventricles has widespread areas of destruction and if, under these conditions vagal stimulation depresses stimulus formation in the auricles the automaticity of the specific tissues * in the ventricles will have been abolished and a contraction does not occur or occurs with insufficient rapidity to provide an adequate circulation Anesthetic agents ** have precisely a similar action on the functional response of the specific tissue, and it behaves as though there were widespread areas of nonfunctioning tissue In other words a heart under anesthesia is as vulnerable to vagal stimulation as a heart with diffuse organic lesions would be without anesthesia[16]

Sympathetic Nervous System i e Vagosympathetic Reflex—This reflex involves the afferent fibers of the vagus and the sympathetic nervous system[27] It is probably this reflex which is responsible for both ventricular tachycardia and fibrillation Oxygen want encourages this type of reflex Abnormal amounts of Adrenalin (epinephrine) in the circulation (either as a result of emotional distress prior to the block pain during a blocking procedure administration of this drug in local anesthetic solutions or a combination of these) may also act as the stimulus which sets off the reflex or sensitizes the myocardium to it The work of Burstein[30] helps confirm the existence of this reflex and he cites cases where Adrenalin administered during inhala

* See Footnote page 70

** Article specifically mentions chloroform cyclopropane ethylene and nitrous oxide but local anesthetic agents might also reasonably be included since these may depress the myocardium

less of the anesthesiologist's attentiveness and ability, little time to treat the problem is available.

The term "cardiac failure" as used in this chapter includes both (1) cardiac arrest which is by far the commonest and (2) ventricular fibrillation which is comparatively rarer. According to Leeds[16] ventricular fibrillation occurs in only 10% of cases of sudden cardiac failure. The recent interest in acute cardiac arrest and ventricular fibrillation is due to the increasing awareness that they do happen and that if recognized early and treated correctly a number of patients may be resuscitated with no clinical evidence of untoward effects from cardiac failure.[1, 77]

Studies on the incidence of cardiac arrest and ventricular *fibrillation from or following* regional block procedures exclusively were not found in medical literature since most articles deal with cases involving all types of *anesthetics. Most sudden cardiac failures re*ported to date have occurred during inhalation or intravenous anesthesia and very rarely as a complication of regional block analgesia per se. The incidence of *such* complications is approximately 1 in 6 000 to 7 500 anesthetizations, regional and general combined.[2, 3] While the incidence of *cardiac failure* as the primary cause of death following regional nerve block cannot be accurately determined, it may be approximately indicated by the following: Bonica[6] reports only two deaths attributable to the block procedure per se following 34 900 regional block cases over an eight year period, *and at the Mason Clinic* two cardiac failures with one death have occurred in 19 000 regional block procedures in the past seven years. Neither Bonica's series nor the Mason Clinic's series include cases of topical application of local anesthetic drugs for cystoscopy, bronchoscopy or *esophagoscopy*; at the Mason Clinic in the past seven years no deaths whatsoever have occurred during these procedures. Since only four cardiac *failures* occurred in 53 900 cases (Bonica plus author), i.e. 1 in 13 475 cases, the incidence of cardiac failures following regional block is probably less than the incidence of such failures following inhalation *or intravenous* anesthesia. This *statement* is further substantiated by the surveys of Beecher and Todd,[66] Cole[11] and Dripps and Vandam.[67] The higher incidence of cardiac *failure following inhalation and intravenous* anesthesia is explainable since agents used in these methods usually have a greater depressing action on the specific tissues* of the heart than do *local anesthetic agents provided* these local agents are not applied directly to the heart or reach it in an extremely high concentration following an inadvertent intravenous injection.

Even though the instances of cardiac failure are few during regional block they may occur *during the surgery* or in the immediate post block *period* as well as during the block procedure itself. Their occurrence during or after therapeutic and diagnostic blocks should warn us that when they occur *during surgery* under regional anesthesia it is not necessarily the surgical procedure that is responsible. In none of the four cases of cardiac failure cited above had surgery been undertaken. The only patient who was to have surgery had been prepared for a cholecystectomy with an epidural block and was in the operating room *when the arrest occurred. Treatment* was immediately instituted but effective manual systole was not established for five minutes and although the patient was resuscitated, *she suffered some mental* impairment. At the present time, one and one half years later, she shows steady progress but has not and probably will not regain complete mental competence. In the other three cases the cardiac failure developed secondarily to an unrecognized and inadequately treated pneu*mothorax* which was *associated with hypoten*sion and oxygen want.

* The term "specific tissue" refers to those tissues of the myocardium which, while difficult to distinguish from the other muscular tissues of the heart by appearance, have the capacity to form stimuli inde*pendently and autonomously.* This is the outstanding differentiating feature of the specific tissue.

the accompanying hypotension and hypoxia invite these more serious consequences.

Age.—An analysis of 1,100 cases of cardiac failure revealed that 207 of all cases occurred in patients under 10 years of age.[31] Incidence declined abruptly above this age and increased again at the age of 40 and above. The high incidence in children can probably be attributed to their highly sensitive reflexes.[34] And it seems that the incidence in the older age group is disproportionately high because changes in the vascular tree of the heart and/or lesions in the myocardium, especially in the specific tissue, render the heart increasingly vulnerable to vagal stimulation.[34]

Position.—Rapid positional changes, the prone and decubitus positions, raising the gall bladder rest, and flexion of the table tend to cause hypotension and narrowing of the pulse pressure with some degree of hypoxia. The lowering of the blood pressure may be due to reflex stimuli or the rapid shunting of blood. Some of these positions also restrict respirations. Therefore, if possible, extremes of positions and rapid turning should be avoided. The latter is of particular importance in the patient with cardiovascular disease.

Vitamin B Complex Deficiency and Endocrine Imbalance.—It has been pointed out that electrocardiographic changes from vitamin B deficiencies occur and that the climacteric is often associated with symptoms related to the heart.[16, 29] Therefore, since these deficiencies and hormone imbalances may contribute to the sensitizing of the heart, it is wise to correct them prior to anesthetization.

SIGNS AND SYMPTOMS

As noted previously, cardiac failure may be the result of primary hypotension and secondary oxygen want or vice versa. In either instance ample warning of the impending cardiac failure is often available to the conscientious physician. For signs and symptoms which precede cardiac failure in such instances see Chapter 4, page 45, and Chapter 17, page 160.

On the other hand, cardiac failure of the *reflex* type occurs suddenly without any such warning signs and symptoms or with only momentary ones. The signs and symptoms of stoppage of the heart in these cases are as follows:

Cadaveric Appearance.—A cadaveric appearance accompanies sudden reflex stoppage of the heart because the peripheral circulation is halted immediately.

Sudden Disappearance of Blood Pressure.—A suspected absence of the blood pressure and the pulse in an extremity should be checked by direct palpation of the large arteries. In case of shock, only, a blood pressure and pulse are often unobtainable in an extremity but pulsations in the carotid artery and aorta are perceptible. In cardiac failure the sudden disappearance of the blood pressure and pulse in an extremity is also attended by the cessation of pulsations in the carotid artery and aorta.

Cessation of Respiration.—When cardiac failure occurs, apnea results. Often during manual systole a gasping type of respiration is seen which presumably represents an effort on the part of the respiratory center to re-establish respirations, even though the heart beat has not been started. This phenomenon is not a signal for elation: in the two cases under inhalation anesthesia in which this was observed by the author, the heart beat could not be re-established, even though the surgeon was working in the thorax at the time ventricular fibrillation began and he immediately instituted manual systole and other restorative therapy.

Dilatation of the Pupils.—The pupils dilate rapidly upon failure of the heart. If effective manual systole is instituted and the patient kept oxygenated, the pupils will contract. If in spite of this therapy the heart beat cannot be re-established, the pupils will again start to dilate. This usually indicates that attempts to resuscitate the patient will fail.

PROPHYLAXIS

Prior to all surgery, but particularly elective surgery, a careful history and physical examination and essential laboratory work (i.e.

tion anesthesia has produced cardiac irregularities and sudden death

Oxygen Want (hypoxia or anoxia)—Although oxygen want itself may cause cardiac failure it is difficult to assess the role it plays in this sudden reflex type Reid *et al*[16] summarize the role of oxygen want under these circumstances when they state "The precise role of anoxia appears to be that while it alone cannot cause a vagovagal reflex nevertheless it potentiates the action of the anesthetic agent so that the net result is further decrease in the capacity of the specific tissue to form stimuli

Depression of the Myocardium by Local Anesthetic Drugs and/or Previous Myocardial Damage—Either individually or together these factors may act to depress conductivity and responsiveness of the specific tissue of the cardiac musculature and thus contribute to sudden cardiac failure (see Chapter 1 page 16) It is known that local anesthetic agents (with the exception of cocaine) have a depressing action on the myocardium and may therefore decrease the normal functional response of this organ If in addition to the depression of the cardiac muscle hypoxia has resulted either from pre existing disease i e arteriosclerosis etc or from the complications of regional block procedures the stage is set for the appearance of cardiac failure (see Oxygen Want above) These factors alone may precipitate sudden cardiac failure but if in addition to these manipulations produce vagal stimulation the chances of sudden cardiac failure are enhanced Nevertheless even though most local anesthetic drugs do have a depressing action on the heart the incidence of cardiac failure during regional block would indicate that the action of local anesthetic agents is not as likely to bring about cardiac failure as is the depressant effect of general anesthetics

Surgical Manipulation—If surgery is begun after a regional block procedure but prior to the *establishment* of the analgesia or if the surgeon starts to work in an unanesthetized area undesirable reflexes of the autonomic nervous system may be initiated Then too pain may cause apprehension and excitement which increase the output of the adrenal gland and this alone or together with the *Adrenalin (epinephrine) in the local anesthetic* solution may markedly increase the *total* circulating adrenaline This is particularly dangerous in a patient with cardiac disease since the combination of the disease excess adrenaline and depression of the myocardium by the local anesthetic agent may result in sudden cardiac failure This may be the explanation of the deaths reported during tho racic sympathetic blocks for angina pectoris[1,6,31] If pneumothorax with its attendant hypoxia is added to the above factors the situation becomes singularly precarious

Some thoracic procedures are at the present time being performed under regional block and in these cases the surgeon should avoid traction torsion and pressure to the heart or *large vessels Such surgical trauma may* trigger cardiac failure Excessive stimulation which may result in the vagal reflex phenomenon of the autonomic nervous system may also originate in the respiratory or gastrointestinal tracts from mechanical contrivances such as endotracheal and Miller Abbott tubes as well as from the clamping or tying of structures containing vagal nerve endings[10]

Diodrast—The use of Diodrast in therapeutic and diagnostic block procedures to assure correct placing of the needle is increasing in popularity Nevertheless a risk is involved severe cardiovascular complications and deaths are reported following its use intravenously and it is always possible to make an inadvertent intravenous injection during a regional procedure[32]

Analeptics—The use of analeptics during *a systemic toxic reaction to a local anesthetic* drug is to be condemned because analeptics increase the oxygen demand of tissues already subjected to hypoxia[33] In this fashion they may help to precipitate cardiac failure

Cerebral Vascular Accidents Coronary Occlusion Pulmonary Embolism, Acute Pulmonary Collapse—These sudden acute accidents may occur during regional block procedures and while they may not cause immediate cardiac arrest or ventricular fibrillation

first While the specific treatment of the fibrillating heart differs from that of the heart in asystole the steps of establishing a tentative diagnosis of exposing the heart and of the artificial re establishment of circulation and respirations all of which precede such *specific* treatment are the same regardless of whether arrest or fibrillation has occurred

Determining Diagnosis and Establishing Artificial Circulation and Respiration —*Alert the Surgeon and Operating Room Personnel* —A conscientious alert physician who is carefully attending the patient, i e checking blood pressure pulse, and respirations following a regional block will be in most instances *the first to suspect a cardiac failure* Additional help must be summoned immediately, and if a surgical procedure is in process the surgeon should be immediately alerted While the anesthesiologist checks for pulsations in the radial and carotid arteries the surgeon should check the aorta or heart if the abdomen or chest is open When the abdomen or chest is not open the only method of conclusively determining whether cardiac stoppage or ventricular fibrillation exists is by means of the electrocardiograph Unfortunately this machine cannot be connected to every patient prior to every regional block procedure for obvious reasons of practicality and since it would take longer than 3½ minutes to obtain an electrocardiogram without prior preparation the instrument except in the occasional case is of little value in confirming a tentative diagnosis of cardiac arrest or ventricular fibrillation Likewise placing a needle into the heart to see if the needle s hub fluctuates—indicating cardiac action—is not too practical since it consumes valuable time and may not give a conclusive diagnosis Placement of a needle in the heart may be done while waiting for the instruments with which to perform a thoracotomy but it is no substitute for direct visualization of the heart *When cardiac failure is suspected a thoracotomy is required*

Such a decision is not always an easy one in regional block procedures particularly if surgery has not started or if the block was performed for diagnostic or therapeutic reasons In such instances Sadove[36] advocates giving intravenously a mixture of 1/75 to 1/50 (0 9 to 1 2 mg) gr of atropine and 0 3 cc of 1% solution (3 mg) of Neosynephrine If no response is then noted within 15 seconds i e , an obtainable blood pressure and pulsations in the arteries, he feels that the immediate institution of manual systole is mandatory Primrose[37] suggested the use of amyl nitrite and states, The ordinary faint recovers immediately with an artificial inhalation of a capsule of this drug whereas the arrested heart cannot respond at all " Striking a sharp blow on the chest wall over the precordium or *passing an electric current through the chest* wall has been employed by Roberts *et al*[38] and Zoll *et al*[39, 40] respectively, as methods to be used to start the heart beating in instances where thoracotomy is not feasible Such a situation may exist during a therapeutic or diagnostic block in an office which lacks additional help and the equipment to perform a thoracotomy However, if the block is performed in an anesthesia block room or the operating room of the hospital where facilities for manual systole should be available these other therapeutic measures seem to be permissible only while preparations are being made for institution of manual systole they should in no way delay thoracotomy once the tentative diagnosis of cardiac failure has been made for thoracotomy is usually the only practical means to a definite diagnosis and effective therapy

It must be recognized that the physician who realizes that time is the essence of treatment *if lives are to be saved* will occasionally perform a thoracotomy in a patient where the heart has not stopped beating and where manual systole is unnecessary While this may be a bit embarrassing for the physician it is something which if it is later carefully and adequately explained to the patient should be accepted without untoward repercussions

Have One Physician Only Give the Orders —It should be obvious that one physician must assume control of the situation when

blood count hemoglobin determination etc) should be performed Nutritional deficiencies call for the administration of vitamin B complex and patients in the climacteric period exhibiting signs and symptoms of cardiac disease should be treated with the appropriate hormone [16, 9] Patients with organic cardiac disease should be well digitalized or quinidized as indicated Fluid and electrolyte balance as well as blood volume should be within normal limits prior to surgery Blood lost during surgery should be replaced *as it is being* lost

Correct premedication is essential and should be carefully chosen for each individual to allay apprehension and depress vagal function Reid *et al* [16, 31] stress the usefulness of atropine in depressing the vagus and their strong belief in its usefulness in this regard is evident in their conclusion In summary one can say that cardiac arrest is a surgical emergency in which time is of the essence Action is imperative However regardless of how successful or spectacular the most ingenious and technically perfect surgical procedures have been or may be surely it must be a sobering thought to realize *that particular emergency was preventable* and preventable by such an old safe and reliable therapeutic agent as *atropine* the very simplicity of which has contributed to its surprising and inexcusable neglect" [31]

The administration of Novocain (procaine hydrochloride) or Pronestyl (procaine amide) *prior to and during the surgical procedure to* decrease myocardial irritability when regional blocks are used is questionable Johnson and Kirby [3] think it is of little protection even during general anesthesia

Perhaps the most important of all prophylactic measures to prevent cardiac failures is to assure maximum oxygenation of the myocardium While hypoxia, per se will not cause or initiate a vagovagal reflex it does enormously potentiate the possibility of such a reaction This is one of the important reasons why the administration of oxygen during regional procedures is strongly favored at the Mason Clinic particularly for patients with a poor physical status and for those who receive a spinal block, an epidural block or intercostal splanchnic (celiac plexus) block —procedures known to cause hypotension and oxygen want

All regional procedures even simple local *infiltrations should be supervised by trained personnel preferably an anesthesiologist* who constantly checks the patient s physical condition

TREATMENT

If the treatment of cardiac arrest or fibrillation is to be successful a rapid diagnosis and institution of manual systole (cardiac massage) is essential otherwise any other type of therapy is no more than an exercise Weinberger *et al* [30] studying the survival time of the brain have shown experimentally that (1) the brain subjected to less than 3½ minutes of anoxia will show no change (2) if subjected to more than 3½ *minutes* but less than 8¾ minutes of anoxia, irreversible changes in the brain occur which are compatible with life but the patient will be mentally incompetent and (3) if more than 8¾ minutes elapse changes occur in the brain which are incompatible with life In this last instance *the heart beat may be re established only to stop two or more hours afterwards*

The exact treatment of cardiac failures may differ from institution to institution and from physician to physician particularly since its exact etiology *has not* been definitely determined However most papers on cardiac failure strongly advise that every hospital maintain surgical instruments syringes endotracheal tubes laryngoscopes oxygen and drugs immediately available and suggest that this equipment be kept separate to be used *only* when this emergency arises

The following treatment is an attempt to summarize the current therapy advocated for cardiac arrest and ventricular fibrillation These therapeutic measures must be instituted simultaneously if possible and the order in which they are listed below is no particular indication as to which should be performed

by checking the blood pressure of the arm. Adequate manual systole should raise the systolic pressure to approximately 60 mm Hg. If it is difficult to obtain this amount of pressure, the aorta distal to the origin of the left subclavian artery may be compressed by a clamp. This will increase the blood flow to the coronary and cerebral vessels as well as allow for a check of the adequacy of the systole by the blood pressure cuff on the upper extremity.[21, 23, 68] Whether the manual systole is carried out by one hand or two hands depends on the size of the heart.[23] If the heart is small, one hand may be effective, but if large, both hands are needed. Whether one or both hands are used, manual systole is tiring and the physician must be relieved at intervals by an assistant if effective systole is to be maintained.

The initial task is to prevent the heart muscle and the brain from dying, not to restore the beat. The beat may usually be re-established unless the tissues which are responsible for initiating it are damaged by hypoxia or anoxia.

Whether the pericardium should or should not be opened is immaterial, provided adequate systole is maintained. However, there is little question that the thoracic approach to the heart is preferable to an abdominal approach because the thoracic approach permits (1) the heart to be seen, (2) the defibrillator to be used, (3) intracardiac administrations to be made, and (4) it is difficult to grasp the heart via the abdomen.[1, 22, 23, 41, 43] Nevertheless, if the abdomen is open at the time of failure, the subdiaphragmatic avenue for manual systole should be used while the chest is being opened.

The use of atropine to decrease vagal reflexes once cardiac failure is *definitely* established is debatable; probably it does no harm.

Intravenous Fluids.—Solutions such as 5% dextrose in distilled water, plasma, blood or other volume expanders should be administered intravenously.[15] They support circulation and increase the return of venous blood to the heart. Sodium chloride solutions are best avoided, as they may increase cerebral edema should resuscitation of the heart be successful. Saklad[44] suggests the use of intra-arterial transfusion if the blood pressure remains low "*because of the improved coronary* and cerebral blood flow it provides."

Avoid Steep Trendelenburg Position.—Seven to 10 degrees of Trendelenburg position may be advantageous to get the blood to the brain, but a greater degree is of no value. It should be remembered that while the head-down position may encourage flow to the brain, it also retards return flow from the brain to the heart, so that perhaps no tilt of the table is the best position for this type of emergency.[4]

Avoid Analeptic and Sympatholytic Drugs.—In cardiac failure, caffeine, Metrazol (pentylenetetrazol), Coramine (nikethamide), ephedrine, Neosynephrine (phenylephrine), etc., should not be used, as they increase oxygen demand and thereby increase tissue anoxia and thus only aggravate the problem. Schmidt[33] writes: "Direct studies of the oxygen consumption of the brain in situ have shown that cerebral metabolic activity runs parallel to cerebral functional activity and that convulsants may lead to cerebral anoxia (with all its consequences) because they increase oxygen demand beyond the available supply."

Avoid Digitalis, Ouabain and Strophanthin.—These drugs act on the auriculoventricular conduction system to increase the refractory period and to slow the rate of conduction and are therefore not indicated in cardiac failure.

Specific Treatment of the Cardiac Arrest (Asystole).—Once the circulation and respirations are maintained artificially, thought may be given to restoring the heart beat. Often these two actions, i.e., establishment of circulation and oxygenation of the tissues, will start the heart's beat. However, if they do not, there is no need for haste and careful consideration may be given to other methods.

Mechanical.—Pricking of the heart or flicking it with the finger may cause the heart to start beating. If these simple methods fail, then drugs are used.

Drugs.—The usefulness of Adrenalin (epi

this complication occurs He alone should direct the treatment Orders by all physicians present will only cause confusion and delay the resuscitation of the heart

Keep Time and Accurately Chart Proceedings—When cardiac failure is suspected the anesthesiologist or preferably some other person, a nurse or intern should immediately be assigned the task of counting *aloud* the passing of time—30 seconds 1 minute 1½ minutes etc This keeps the physicians involved in the active treatment acutely aware that speed is essential The time keeper should also accurately chart *each* step i e intubation manual systole drugs administered etc as it is performed by the surgeon and anesthesiologist

Remove Cause of Failure—If surgical instrumentation of the organs is under way or a block procedure is being performed it must cease immediately so that additional stimulation is obviated

Discontinue Anesthesia Clear Airway Correct any Possible Hypoxia—If inhalation or intravenous anesthesia is being used along with the regional procedure it must be discontinued at once An adequate airway using an oral airway or preferably a cuffed endotracheal tube should be established A cuffed endotracheal tube is favored because it prevents (1) aspiration of vomitus and (2) when artificial respiration is given by bag and mask it prevents the dilatation of the stomach which in itself may cause death If only an oral airway is used, the physician must watch for distention of the stomach and if it occurs a Levin tube for its decompression must be passed

Should vomiting occur the pharynx larynx trachea etc must be cleared by suction (see Chapter 18 page 167) Bronchoscopy is the most satisfactory means of accomplishing this When the airway is clear artificial respiration at the rate of 20 per minute preferably by bag and mask using 100% oxygen at 10 liters per minute should be maintained Artificial respiration may by the very nature of the recoil and expansion of the lungs compress the heart and send some blood to the coronaries and the brain but it is *never* a substitute for manual systole

If oxygen bag and mask are not available immediately, manual types of artificial respiration and/or mouth to mouth breathing may be used until they are Correction of oxygen want is essential

Institute Manual Systole (cardiac massage)—This is by far the most important step in the treatment of cardiac failure and must be instituted within 3½ minutes after the interruption of circulation to the brain

Preparation of the skin and sterile draping of the thorax is an unnecessary waste of time In the unprepared skin over the left fourth or fifth interspace a slashing deep incision should be made and rapidly extended down to the pleura avoiding the internal mammary artery if possible The pleura is opened by the knife handle so as not to cut the lung and the lung depressed so that the pleura may be widely opened with scissors or a scalpel If however the lung is cut immediate repair must not be attempted—only a clamp should be placed across the rent It can be sutured later Once the pleura is opened the right *hand is forced between the ribs and the heart* is palpated *No more than 15 seconds should elapse between the time the scalpel is handed to the physician and the hand is on the heart*"[3] If the heart is not beating effectively irrespective of whether asystole or ventricular fibrillation is found manual systole at a rate of 60 to 80 per minute is started The thoracic cage is then opened wide by an assistant or by the surgeon using his left hand as the right continues the manual systole To accomplish this the costal cartilage of the ribs is usually cut and a self retaining retractor inserted This allows direct visualization of the heart and makes the manual systole easier as it relieves the pressure of the ribs on the wrist or forearm of the inserted hand

The term "manual systole is preferred to cardiac massage because rhythmical compression is of no value unless it is performed in a manner which forces blood from the heart and simulates an effective systole The effectiveness of the systole should be ascertained

(procaine) should not be used intravenously or intracardially in cardiac arrest for they would further depress the myocardium.

Specific Treatment of Ventricular Fibrillation.—This is a very serious problem since the heart must be converted to asystole (cardiac arrest) before the normal beat can be restored. When this complication occurs in a normal heart, resuscitation is usually possible, but when it occurs in an organically diseased one, it is almost always fatal. *No attempts to stop the fibrillation should be made before effective respirations and circulation have been established by artificial means* as described previously to assure adequate oxygenation of the tissues, particularly of the myocardium. Once these are established, the following means to stop fibrillation and reestablish the heart beat may be undertaken.

Defibrillation of the Heart.—If manual systole in itself has not brought about defibrillation, then the heart may be defibrillated either by electrical methods or by drug therapy.

To effect defibrillation of the heart by electrical shock methods, two electrodes are applied to the ventricle (one at the back and one at the front opposite each other if possible) and 125 to 130 volts at a standard 1.5 *to 3.0 amperes are passed through it for* 0.1 second. Stronger voltage and/or periods of application longer than 0.1 second may burn the heart.[1, 73] If the fibrillation is not stopped, then another shock may be administered.[57, 1] A period of manual systole should be instituted between single shock trials to assure adequate oxygenation of the brain and heart muscle. The injection of 2 to 5 cc. of 5% Novocain (procaine) into the right ventricle after the first unsuccessful shock may also make the heart respond more favorably to subsequent shock therapy.[54] The experimental work of Kay[49] does not substantiate the efficacy of using Novocain in ventricular fibrillation. He states: "The experimental results here recorded tend to indicate that procaine should not be used unless serial defibrillation has failed after many attempts. Although the procaine appeared to be of slight assistance in electrical defibrillation of the heart, the ventricular standstill that may result following successful electrical defibrillation was more difficult to treat if 10 cubic centimeters of 1% procaine had been used, and in most experiments this disadvantage appeared to outweigh the advantage in the use of procaine."

Should single shock therapy be ineffective, then a series of 5 to 10 shocks at 0.1 second intervals using the same voltage and time of application should be given as the electrodes are moved over the ventricles.[49, 5] Moving the electrodes is necessary to avoid burning the heart as well as to cover all areas of the heart from which the stimulus causing the fibrillation may be originating. Many investigators feel that serial shocks are more effective than the individual shock technique in stopping fibrillation.

Defibrillation by drugs may at times be more effective than mechanical defibrillation. However, this is debatable.[49] The physician may, so to speak, pay his money and take his choice. Often it is necessary to use both methods to bring about asystole. The following drugs are effective defibrillators and one or more may be tried by injection into the chamber of the auricle or ventricle (see Table XIV, page 78).

I. Novocain (procaine hydrochloride).—10 to 20 cc. of a 1% solution.

II. Pronestyl (procaine amide).—100 to 200 mg.

III. Potassium chloride.—0.5 to 2.5 milliequivalents (1 to 5 cc. of a 3.7% solution).

IV. Xylocaine (lidocaine hydrochloride).—Recently this drug has been shown to correct ventricular fibrillation effectively in 21 out of 23 dogs.[69] These preliminary experiments showed that ventricular injection of Xylocaine was the most effective treatment for ventricular fibrillation in dogs. As yet studies concerning the use of this drug in man are not available. It can be as

TABLE XIV

DRUGS THAT ARE USEFUL IN CARDIAC RESUSCITATION
(*After Leeds*[15])

Drug	*Dosage*	*Indications*	*Contraindications*	*Remarks*
Adrenalin (epinephrine)	0.5 to 2 cc 1:5,000 solution in auricle	Regular rhythm weak contractions cautiously in asystole	Ventricular fibrillation	Stimulates heart by direct effect on myocardium and conduction system increases myocardial irritability
Calcium chloride	2 to 4 cc 10% solution in left ventricle	Regular rhythm weak contractions cautiously in asystole	Ventricular fibrillation	Will sometimes revive heart that does not respond to epinephrine
Novocain (procaine hydrochloride)	10 cc 1% solution in auricle or 50 to 100 cc 1% solution intravenously	Ventricular fibrillation that fails to respond to shocks	Asystole and flabby myocardium	Diminishes irritability of myocardium and conduction system
Pronestyl (procaine amide)	100 to 200 mg in auricle or intravenously	Ventricular fibrillation that fails to respond to shocks	Asystole and flabby myocardium	Diminishes irritability of myocardium and conduction system
Atropine and scopolamine	0.0065 gm or 0.01 gr intravenously	Cardiac arrest due to vagovagal reflex	Tachycardia	Block vagal effects on sino-auricular pacemaker
Quinidine	0.2 gm 4 or 5 times times daily orally	Presence of cardiac arrhythmia preoperatively	Intravenous or intracardiac use	Decreases myocardial excitability and slows conduction of impulses
Barium chloride	1 to 2 cc 0.5% solution intracardially	Regular rhythm weak contractions cautiously in asystole	Ventricular fibrillation	Used less frequently than calcium chloride
Potassium chloride	0.5 to 5% solution intracardially	Ventricular fibrillation to produce its inhibition	Asystole or weak regular contraction	Not recommended electrical defibrillation is probably more efficient and less dangerous

nephrine) has been discounted by Primrose[3] but supported by a number of other authors[1, 14-46, 49] It may be given in small doses i.e. 0.5 to 2 cc of 1:5000 into the auricle or ventricle Larger doses may cause ventricular fibrillation (see Table XIV page 78)[47] It should not be given before oxygenation of the heart muscle has occurred from at least one minute of effective manual systole lest ventricular fibrillation ensue

Calcium chloride has proven to be very useful and 2 to 4 cc of a 10% solution injected cautiously into the chamber of the left ventricle is usually an adequate dose but if it is not the dosage may be repeated (see Table XIV page 78)[49, 50] The chloride is important because only an ionizable calcium salt will prove effective Calcium chloride must be used judiciously for if too much of this drug is injected into the left ventricle fibrillation may result

Pronestyl (procaine amide) and Novocain

(procaine) should not be used intravenously or intracardially in cardiac arrest for they would further depress the myocardium.

Specific Treatment of Ventricular Fibrillation.—This is a very serious problem since the heart must be converted to asystole (cardiac arrest) before the normal beat can be restored. When this complication occurs in a normal heart resuscitation is usually possible but when it occurs in an organically diseased one it is almost always fatal. *No attempts to stop the fibrillation should be made before effective respirations and circulation have been established by artificial means* as described previously to assure adequate oxygenation of the tissues, particularly of the myocardium. Once these are established the following means to stop fibrillation and reestablish the heart beat may be undertaken.

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sumed that it can be used in a dosage similar to that of 1% Novocain i e, 10 to 20 cc of a 1% solution

Re establish heart beat—If defibrillation is effective then attempts to re establish heart beat as described under Cardiac Arrest page 77 to 79 must be undertaken

Precautions—Once the heart has started to beat normally the following precautions should be observed

Do Not Close the Chest Too Soon—The chest must not be closed prematurely for cardiac failure may recur [9]

Do Not Do Additional Surgery—When cardiac failure occurs and it is treated satisfactorily other surgery *unless in itself life saving* must be postponed to another time It is better to stop surgery and let the patient live to be operated on another day than to have him die trying to overcome the insurmountable odds of cardiac failure plus a long surgical procedure

> For he who fights and runs away
> May live to fight another day
> But he who is in battle slain
> Can never rise and fight again"
>
> —Oliver Goldsmith [71]

AFTER CARE OF THE PATIENT RESUSCITATED FROM CARDIAC ARREST OR VENTRICULAR FIBRILLATION

Once a patient with ventricular fibrillation or cardiac arrest has been resuscitated the physician may draw a *small sigh of relief* But he should not be over optimistic for if postoperative treatment is not carefully planned and supervised the patient may die from the effects of cerebral edema caused by the anoxia which is a result of acute cardiac failure Saklad [44] states "After re establishment of the heart beat any one of three things may result There may be complete recovery of the heart beat respiration and cerebral function The *heart may function* properly respiration may be satisfactory but because of advance oxygen deprivation to the brain there may be altered cerebral *function* On occasion the heart may recover but respiration will not be resumed with ultimate death of the patient Often a patient may linger unconscious but breathing well with *seemingly good heart action and die after a* protracted period of 1 to 15 days These patients exhibit the characteristic brain damage of cerebral hypoxia"

Seldon *et al* [56] stress the importance of the relationship of cerebral edema in the post operative period to cerebral anoxia during the surgical period and state *Permanent damage to the brain as a result of acute cerebral anoxia not only is a function of the duration of the anoxic episode but also in the case of sublethal periods of anoxia must be conditioned by the duration and severity of post anoxic cerebral edema* It is suggested that a considerable portion of the permanent cerebral damage reported in these unfortunate cases may be the result not of the initial insult but rather a period of untreated cerebral edema subsequent to the initial insult"

Courville [67] Saklad [44] and Lucas [9, 9] have found that the earliest change in the anoxic brain is edema If this is rapidly absorbed the cells recover but if it persists then further oxygenation of the cell is impaired by purely mechanical means so that hypoxia of the cell is aggravated and a vicious cycle is established that eventually leads to the death of the cell *The signs and symptoms of brain* edema are referable to the central nervous system i e *coma restlessness screaming convulsions etc* When these are seen the *outcome is questionable* Treatment to reduce cerebral edema must be immediately instituted with other generalized supportive therapy if these patients are to be saved from brain damage and death

The physician should not wait until the above signs and symptoms develop As soon as the patient is returned to the ward the physician must institute the following therapy which is aimed at lessening the cerebral edema *oxygenating the brain cells and reversing the vicious cycle*

Administer Serum Albumin — Although *serum albumin constitutes less than 60% of* the total plasma proteins it is responsible for

about 80% of the total osmotic effect of plasma.[illegible] Fifty cc. of albumin is osmotically equivalent to 250 cc. of plasma and will usually withdraw 3 to 3½ times its own volume of fluid from the tissues in fifteen minutes.[61] Gates and Craig,[60] Seldon *et al.*,[illegible] Shelden *et al.*[illegible] and Moyer[illegible] believe that serum albumin is superior to most of the commonly used dehydrating agents, such as 50% dextrose or sucrose and other blood volume expanders. Coles'[64] case of cardiac arrest which exhibited evidence of cerebral edema in the postoperative period and which was successfully treated with serum albumin would substantiate this preference. However, if serum albumin is not available, the other solutions may be used.

Dosage.—Gates and Craig[60] advocate that 60 to 80 cc. of serum albumin solution (5 gm./20 cc.) be administered intravenously in 6 to 10 minutes and that fluids be withheld from the patient for the next 8 hours. Doses as large as 300 cc. to 500 cc. (75 to 125 gm.) of salt poor human serum albumin have been given over a one hour period without untoward effects to reduce cerebral edema associated with barbiturate poisoning.[illegible] Cases of cardiac failure at the Mason Clinic in which serum albumin was used demonstrate that 80 to 120 cc. (20 to 30 gm.) can be administered intravenously in 6 to 8 minutes every 4 to 6 hours without harm.

Complications of Administering Serum Albumin.—None have been observed at the Mason Clinic, but Cutter mentions pulmonary edema, respiratory embarrassment and cardiac decompensation as possibilities.[61]

Restrict Intravenous Fluids.—During treatment of cerebral edema fluids should be withheld or limited to a minimum. Normal saline solutions should be avoided under most circumstances as they may cause death by accentuating the edema.

Administer Oxygen.—Oxygen should be administered in high concentration by mask or tent.

Use Antibiotics.—These patients may develop pneumonia. If the chest wall has been entered without using asepsis, as is likely, the chest wall, the pleural cavity and/or the pericardial space may become infected. All precautions to avoid infection must be taken.

Supportive Therapy.—High vitamin intake, food and fluid by mouth, stir up regime (tracheal suction, moving from side to side, coughing of the patient, and early ambulation), good nursing care, and effective means to reduce hyperthermia if present, i.e., alcohol rub, ice packs, etc. will all contribute greatly to the patient's recovery.

Care for Chest Wound.—The usual routine care of a chest wound, i.e., under water drainage, etc., will suffice.

COMMENT

The Intravenous Use of Novocain (procaine hydrochloride) in Cerebral Edema.—The use of intravenous Novocain to overcome the acute anoxic state following episodes of cerebral anoxia has been advocated by Courville.[1] He states: "The encouraging experiences with the use of procaine hydrochloride (1 gm. in liter of 2% glucose in normal saline) in the treatment of cerebral anoxia of other causes (Olsen, Amyes, and Marinacci, 1950) would suggest its value in these postanesthetic cases. Its use is indicated in view of the secondary and often long standing vasomotor effects of anoxemia already discussed. Procaine is presumed to stabilize vasomotor control with consequent improvement in the patient's clinical state if the original insult has not been too profound. It should be given intravenously by slow drip once or twice a day until the patient has recovered or until further efforts are obviously useless."

It is our belief that serum albumin is preferable to intravenous Novocain. The use of intravenous Novocain as advocated above necessitates the administration of large volumes of solutions. One of the principles of treatment of acute cerebral edema is the restriction of fluid intake.

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CHAPTER 8

Pain

This chapter is a general survey of the causes of pain which may be associated with a regional block procedure Pain accompanies many complications discussed in this text and in most instances it is of concern only in that it causes the patient discomfort However it may be a signal of a serious complication e g pneumothorax neuritis spinal cord injury etc and chapters concerning these complications should be consulted

ETIOLOGY

When pain alone occurs during the execution of a block procedure it is usually caused by minor trauma, but when it persists for more than a few days following a block causes other than the initial trauma should be sought infection tissue degeneration etc In general pain following a regional block procedure is due to

Tissue Trauma—Trauma to tissues from needle punctures alone is of no serious consequence The patient may complain (1) of burning caused by distention of the tissue when a skin wheal is made (2) of electric like sensations when a nerve ganglion plexus of nerves or the spinal cord is impaled and (3) of deep aching pain when the muscles and periosteum are pierced In most of these cases the pain ceases immediately following the injection of the local anesthetic agent or the removal of the needle and if it returns it persists for only a day or two and requires no analgesics for its control

The exception to this is the development of (1) a hematoma under the periosteum (2) an abscess or osteomyelitis (3) neuritis or (4) injury to the spinal cord In these cases the pain may persist for a prolonged period of time particularly if the nervous tissue is damaged Pain from injuries to the spinal cord is usually permanent while that which results from injuries to the peripheral nerves or their roots often disappears as the nerve regenerates However in some cases pain following peripheral nerve damage may persist even after the nerve heals because a pattern of pain is established which perpetuates itself

Injection of Unphysiologic Solutions—Pain may occur following the injection of

Local Anesthetic Agents Dissolved in Distilled Water—Pain from the injection of distilled water usually disappears within a few minutes and does not recur

Neurolytic Drugs—The initial pain which follows the use of neurolytic drugs i e alcohol phenol propylene and polyethylene glycol ether and excessive concentrations of local anesthetic drugs usually disappears as the drug used exerts its anesthetic effect but as this wears off the pain recurs if neuritis sloughs or sterile abscesses develop (see Chapter 10 page 102 and Chapter 11 page 114)

Certain Local Anesthetic Solutions e g Cyclaine (hexylcaine)—Ruben and Anderson [1] report that Cyclaine burns on injection but that this response disappears when the drug exerts its anesthetic effect and usually does not reappear when the drug s action is dissipated Only two exceptions to this have been called to the author s attention In these a brachial neuritis followed the use of this drug to block the brachial plexus

Solutions Contaminated by Metallic Ions Released from Metal Parts of Syringes and Receptacles—Solutions contaminated by metal ions seldom cause pain during the physiological effect of the local anesthetic drug but may do so if swelling of the tissues and/or neuritis develop (see Chapter 10, page 94 and Chapter 11, page 114).

Infection—Pain almost invariably accompanies infection and this is particularly true if osteomyelitis or an abscess results.

Pneumothorax—(see Chapter 6, page 58).

Stellate Ganglion Block—It is not uncommon during the placing of the needle in the region of the stellate ganglion or during injection of the solution into it that the patient complains of pain in the shoulder, in the chest, or in the jaw, particularly if the needle is correctly placed. This type of pain resembles that which occurs in angina pectoris and in a patient with coronary artery disease may represent true angina. Pain in the shoulder, scapula, or chest usually indicates stimulation of the stellate ganglion while pain in the neck and jaw usually indicates stimulation of the cervical sympathetic chain above the region of the stellate ganglion. However, the pain may also be caused by stimulation of the periosteum, soft tissues or somatic nerves. In most cases the pain is due to stimulation of the ganglion itself or other sympathetic fibers when the needle is correctly placed. When the needle is incorrectly placed the pain is due in part to the stimulation of periosteum or bone and this type of pain may be referred to a somatic region.

Vascular Accidents.—Pain (other than angina) accompanying vascular accidents may occur following regional block procedures as a result of the normal "borrowing-lending" mechanism of the vascular system which may follow a block procedure (see Chapter 25, page 235).

Pain in the Legs During Administration of Epidural Blocks—When a large volume of solution is initially injected into the epidural space, particularly in the caudal region, the patient may complain of an aching, cramping pain in the buttocks, thighs and calves. This occurs relatively frequently if the local anesthetic agent is injected rapidly when administering a caudal to a lightly medicated obstetrical patient. It is no sign to discontinue the procedure, although the injection of the local anesthetic solution should be slowed or momentarily stopped. As a matter of fact, this type of pain is one of the most reliable signs that the needle is correctly placed in the caudal canal.

Diodrast—(see Chapter 10, page 101).

SIGNS AND SYMPTOMS

Pain obviously varies in intensity and may be dull, throbbing, burning, sharp, stabbing or a combination of these, depending on its cause. The pain may be more severe during motion or when the physician exerts pressure over the area. In general, pain which accompanies pleural irritation, neuritis, abscess formation, spinal cord injuries and other serious complications is the most troublesome and its relief requires heavy sedation and, in some extreme cases, other nerve blocks with local anesthetic agents.

PROPHYLAXIS

The avoidance of pain during and following regional block procedures requires: (1) correct medication, (2) explanation of the procedure so that the patient does not jump when the needle is placed and thereby cause its misplacement, (3) avoidance of multiple traumatizing placements of the needles, (4) prevention of infection from breaks in technique, and (5) the avoidance of neurolytic drugs unless the patient is cognizant of their possible complications and the physician is willing to accept the risk of a neuritis, of an abscess, etc.

TREATMENT

In most instances pain is of a minor nature and its relief requires only (1) active use of the part involved, (2) local heat, (3) physiotherapy, and/or (4) aspirin (acetylsalicylic acid) 10 to 25 grains. If the pain arises with

sudden severity or if it persists and becomes more severe, then the possibility of the development of a serious complication must be considered The specific treatment of pneumothorax, neuritis slough abscess cellulitis etc, may be found in the chapter specifically devoted to these problems

If anginal pain sets in during a stellate ganglion block and if the needle is thought to be correctly placed the best treatment is the injection of the blocking agent The author feels as do Adriani *et al*[3] that if a patient complains of pain in the chest during needle placement the procedure should be carried to completion and the blocking agent injected The pain caused by the placement of the needle or the stimulation of the stellate ganglion may result in constriction of the coronary vessels but paralyzing the ganglion or the area from which the exciting stimulus originates by injecting the anesthetic solution will usually relieve the pain either by anesthetizing the sensory fibers in the area or by dilating the coronary vessels[4]

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CHAPTER 9

Bleeding Following Puncture of Blood Vessels

THE PLACEMENT of needles during regional block procedures may cause bleeding into the tissues from trauma to a blood vessel. This may only result in ecchymosis or the formation of a hematoma, but under specific circumstances either permanent tissue damage or death can follow.

ETIOLOGY

The primary cause for the bleeding is, of course, the puncturing or tearing of a vessel from either needle placement or removal. Whether or not the bleeding from a damaged vessel produces minor or serious consequences depends on: (1) the anatomical structure of the area of the body being injected, i.e., whether the nerve lies in a space, a potential space or is surrounded by firm mesenchymal tissue; (2) the vascularity of the area being injected; (3) the coagulation time of the blood; and (4) the drug injected.

Anatomical Structure of the Area of the Body Being Injected.—Bleeding into areas of the body other than the face will be confined to small areas of ecchymosis and is usually of no serious consequence in the patient with a normal clotting time. The fascia, muscles, ligaments, tendons and periosteum, as well as the hyaluronic acid in the subcutaneous tissue, act as barriers to limit the spread of blood and the clotting mechanisms seal the vessels. Therefore, large hematomata seldom form.

However, if a block of the branches of the trigeminal nerve is undertaken and a blood vessel is punctured, a large hematoma may form in the eyelids, cheek or jaw within 10 to 30 seconds. The face is particularly vulnerable to this complication because its very pliable, easily stretched skin offers no obstruction to the spread of blood (see Figure 17, page 87). In these cases the bleeding goes unabated until either the blood vessel seals itself or the blood fills the space and the pressure outside the vessel equals the pressure from within. Other than temporary discoloration and distortion, this type of complication



Figure 17. Bleeding following block of the maxillary nerve at the foramen rotundum.



Figure 18 Discoloration and proptosis of an eye following a maxillary alcohol block at the foramen rotundum for tic Acute hemorrhage into the orbit followed the block with gradual loss of vision over a period of 5 minutes Patient could not open his eye lid

causes no problem except in the orbit Here the space is limited and even in the patient with a normal clotting mechanism the bleeding may be rapid enough to cause excessive intraorbital pressure and damage to the optic nerve before the vessels seal (see Figure 18 page 88 and Figure 19 page 89)

Vascularity of the Area Injected —The head and neck are highly vascular in comparison to other parts of the body and therefore the chances of inadvertent puncture of a vessel in these areas are many times greater than in the other body areas In addition the face often reacts to trauma by "flushing i e the vessels dilate and this in itself predisposes to bleeding These factors in addition to those of the anatomical structure of the head mentioned previously account for the high incidence of hematomata following peripheral nerve block or even local infiltration in this part of the body

Coagulation Time of the Blood —When a patient has an extended clotting time due to a blood dyscrasia (hemophilia etc) or anticoagulant therapy and a blood vessel is traumatized a catastrophic hemorrhage may result[1 8] For example In the normal healthy patient bleeding into the epidural subarachnoid or retroperitoneal spaces is not serious because the hole in the blood vessel is closed by the clotting mechanism of the blood before pressure can build up to a degree sufficient to damage a nerve or before a severe loss of blood endangers the patient s life But if the patient s clotting time is longer than normal blocking of the nerves coursing through these spaces is singularly hazardous In such a case if the hemorrhage goes unrecognized or proves to be uncontrollable a neurological complication or death may result " [6]

Drug Injected —Neurolytic drugs i e alcohol phenol Efocaine etc not only cause degeneration of the nerve but also may damage other tissues resulting in ecchymosis even when injected in the patient with a normal clotting mechanism In one case at the Mason Clinic the use of Efocaine in an intercostal block resulted in an ecchymotic area which measured 6 by 10 inches It is debatable whether the Efocaine (Novocain in propylene glycol) caused damage to the blood vessel or whether the Novocain per se by its vasodilatory action prevented the normal physiologic retraction of the vessel and its rapid sealing In any case with aqueous local anesthetic solutions we have not observed such a reaction in over 2000 patients receiving intercostal block

Ecchymosis may also follow the subcutaneous injection of Diodrast This has been observed by the author and one of his residents LeGrande Anderson following the subcutaneous injection of the drug into their forearms to investigate deleterious effects which might appear following the use of this drug in diagnostic or therapeutic block procedures

If the above mentioned drugs are injected into a patient with an abnormal clotting mechanism the resulting hemorrhage will probably be more severe

SIGNS AND SYMPTOMS

The signs and symptoms of bleeding depend on the amount of bleeding and the area or space into which the bleeding occurs. The onset may be recognized immediately by the swelling and other signs, or it may be insidious, with a neurological complication or a severe fall in blood pressure as the first evidence of hemorrhage.

Swelling, Ecchymosis and Pain.—One or more of these is usually the first sign of bleeding into the mesenchymal tissues, the orbit and the retroperitoneal space. One may occur without the others, but in many instances all are present. None but pain is seen when bleeding into the epidural or subarachnoid space occurs.

Hematoma.—If the swelling is marked, a hematoma may have occurred. Hematomata the size of large grapefruit forming in the retroperitoneal space following lumbar sympathetic block in conjunction with anticoagulant therapy have been reported.[5, 6] Graves[6] reported five such cases, in one of which the hematoma was not noted and the correct diagnosis was not established until five days had elapsed. This patient recovered.

We have seen a subperiosteal hemorrhage in the sacral area following transsacral block in a female with a normal clotting time. It forced the patient to remain in bed for 10 days. In this case walking markedly accentuated the pain which radiated into the hip joint and down the leg. Cases of this severity are infrequent.

Only occasionally does a hematoma persist longer than a few weeks or become calcified. Even when hematomata calcify they do not usually cause pain. My associates and I have seen only one instance in which a hematoma did not absorb in less than two to three weeks. In this case a hematoma the size of a chicken egg formed from trauma to the subclavian artery after a brachial plexus block. It persisted without causing pain for six months before disappearing, and during this time the man's only complaint was of swelling. X-ray studies revealed small areas of calcium in the hematoma, but this was eventually absorbed. This complication does not present a contraindication for brachial block procedures, for the case here cited is the only one of its kind to appear in over 1,000 brachial plexus blocks performed at the Mason Clinic.

Neurologic Signs and Symptoms.—Neurologic signs and symptoms seldom occur from bleeding into the tissues, but they may result from bleeding into the orbit and epidural or subarachnoid spaces. Such bleeding is a distinct possibility when the coagulation time is prolonged, but also may happen when it is normal.

Bleeding into Orbit.—Bleeding into the orbit is usually first discovered from a swelling or apparent swelling of the eyelids and a marked exophthalmus. As tension mounts, pain in the eye develops, diplopia ensues, and, if the bleeding does not stop, loss of vision follows within 5 minutes or so from pressure on the optic nerve and/or the ophthalmic blood vessels. In one such case seen at the Mason Clinic the blindness was permanent (see Figure 18, page 88, and Figure 19, page 89).

Bleeding into the Epidural and Subarachnoid Space.—Usually the first signs and symptoms of this complication are motor and sen-



Figure 19. Patient shown in Figure 18, 4 days later. (*Bottom*) Shows decrease in swelling of eyelid and discoloration. (*Top*) Shows ability to open eyelid. Motion of the eyeball and action of the pupil are the same as of the normal eye. However, the patient has not recovered any vision in this eye.



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sory changes, and their onset is usually noted 4 to 24 hours following full recovery of sensory and motor function from the anesthetic procedure A retap of the patient may disclose frank bleeding Bleeding into the epidural space does not usually produce severe or permanent neurologic sequelae since the blood may escape via the intervertebral foramina and marked pressure on the nerves may not develop Bonica[5] reports an instance of bleeding into the subarachnoid space in a patient with a prolonged clotting time in which the first evidence of the complication was a cauda equina syndrome

Blood Pressure, Pulse and Respiration—Normally there is little or no change in the blood pressure following small areas of ecchymosis However hemorrhage into the epidural subarachnoid or retroperitoneal spaces can be of sufficient magnitude for the blood pressure to fall and the pulse and respiratory rates to increase If the cause of these signs and symptoms is not recognized and treated immediately, severe shock may rapidly develop and death may result Of the five cases of severe hemorrhage into the retroperitoneal space following the use of lumbar sympathetic blocks in conjunction with Dicumarol therapy which were reported by Graves[6] two of the patients died from the complication O Connor *et al*[7] also report a fatality from the same cause

PROPHYLAXIS

Do a Careful History and Physical—Patients who are to have a regional block procedure whether for an operation or a diagnostic or therapeutic block are entitled to a complete history and physical examination before the block is executed Otherwise blood dyscrasias etc, may not be noticed until it is too late

Avoid Trauma—The needles used in block procedures should be sharp and the gauge should be small, i e 22 gauge or smaller Multiple punctures should be avoided particularly if a therapeutic sympathetic block is used in conjunction with anticoagulant therapy

Apply Pressure to Bleeding Area—If a blood vessel is punctured manual pressure should be immediately applied over the area so as to compress the soft tissue area into which the blood may flow, thereby avoiding or at least decreasing the size of the resultant ecchymotic area or the hematoma

Avoid Regional Block for Surgical Procedures in Patients with a Prolonged Coagulation Time—The availability of medical anesthesia in most areas and progress in the development of new drugs and gases for general anesthesia have rendered it likely that a correctly administered inhalation or intravenous anesthetic will be tolerated by most patients without ill effect Therefore it is perhaps best to avoid regional block procedures in patients whose clotting time is prolonged

Avoid the Use of Sympathetic Block When Anticoagulant Therapy Has Been Instituted—A number of authors have cautioned against the use of these two types of therapy at the same time[1,8,14] Pratt[9] does not agree with them He believes that the ideal treatment for vascular lesions is the concomitant use of anticoagulant drugs to prevent thrombus formation and emboli and sympathetic block to prevent vasospasm of the damaged vessels as well as reflex vasospasm of its collaterals Furthermore he is convinced that if the physicians involved in the treatment of such a patient are skillful and carefully observe the patient who is receiving both types of therapy the action of the anticoagulant drug may be reversed without complications if hemorrhage should start On this subject he states

It is likely that serious and fatal hemorrhages have been blamed on anticoagulant drugs when lack of skill on the part of the person administering them was the true cause We have been through the phase of dangerous and fatal anesthesia given by untrained interns and nurses Thiopental (Pentothal) sodium or spinal anesthesia will kill in seconds if incorrectly administered Patients have died from tension pneumothorax because someone did not know enough to aspirate the air from the pleura Yet, we do not hesitate

to use these anesthetics or to open the chest We do however demand that the administrator know the anesthetic and how to handle it We also make certain that the postoperative period after thoracic surgery is correctly handled As with other therapeutic modalities the safety of anticoagulant drugs as well as sympathetic nerve blocks used singly or in combination depends largely on the ability and knowledge of the person who is administering them If due precautions are exercised these two complementary forms of therapy can be used together safely for the *benefit of the patient*"

To date at the Mason Clinic we have not hesitated to use sympathetic blocks in a patient receiving anticoagulant therapy We insist on the use of a 22 or smaller gauge needle and avoid multiple traumatic punctures No hemorrhage of a severe nature has been encountered up to the present

Furthermore it should be remembered that when bleeding occurs during the concomitant use of anticoagulant therapy and sympathetic block the puncture of a blood vessel from the block procedure is not always responsible The literature adequately establishes that hemorrhage may occur spontaneously during anticoagulant therapy (see Figure 20 page 91)[4, 10, 11]

TREATMENT

Bleeding is treated according to its severity its cause and the signs and symptoms which appear

Swelling, Ecchymosis, and Hematomata — When swelling is first noted the application of pressure and cold packs over the area may greatly reduce the degree of swelling and avoid the formation of a hematoma If this treatment is unsuccessful and a large area of ecchymosis or a hematoma results the injection of 10 to 20 cc of normal saline containing 150 to 300 T R U of hyaluronidase (Diffusin Wydase Alidase) into and around the hematoma or ecchymotic area followed by gentle massage of the area and a pressure dressing will greatly facilitate the absorption of the blood This therapy decreases the size of the hematoma and causes the ecchymotic area to disappear more rapidly[1, 13] Once the bleeding has stopped warm moist dressings may hasten the reabsorption and also lessen pain should it be present The pain seldom warrants sedation

Orbital Swelling — Usually little can be done about bleeding into the orbit since it occurs so rapidly Nevertheless, whenever this complication results irrespective of its severity consultation with an ophthalmologist should be sought immediately

Bleeding into the Retroperitoneal, Epidural or Subarachnoid Spaces — Treatment in these cases involves (1) the correction of hypo



Figure 20 This patient was referred for a stellate ganglion block with possible *embolism of the brachial* artery A review of her history revealed that she was on anticoagulant therapy following pelvic surgery No block was performed when it was found that clotting time was greatly increased The swelling of the arm was thought to be due to *bleeding* into the tissue This proved to be the case for when the clotting time was returned to normal the arm stopped swelling and improved

tension and shock when they occur (2) the reversal by the internist of the anticoagulant therapy if such is being used and (3) immediate consultation with a neurosurgeon if bleeding into the epidural or subarachnoid space is suspected and/or neurological signs and symptoms develop

Reverse the Effect of Anticoagulant Therapy—It should be noted that when heparin is used and bleeding results the action of the heparin may be cancelled by using whole blood transfusion protamine or both If Dicumarol (bishydroxycoumarin) is used instead of heparin whole blood or vitamin K or both will cancel its action

Correct Hypotension—If bleeding is brisk and a severe hypotension develops the blood pressure must be elevated This is particularly important in the patient with cardiovascular diseases since the hypotension may precipitate other sequelae i e coronary occlusion thrombosis in other parts of the body etc

If whole blood is not immediately available vasoconstrictor drugs or blood volume expanders may be used to raise the blood pressure However these are no substitute for a blood transfusion when hypotension is the result of hemorrhage

Ligate Bleeding Vessel—It is possible that in certain cases surgery and ligation of the bleeding vessel may be necessary However the literature reviewed has not cited such a case

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Local Toxic Reactions to Drugs Used in Regional Block

(*Swelling, Cellulitis, Abscess, Gangrene and Slough*)

The term "local toxicity" is defined as a reaction of the tissue at the site of the block. Swelling, cellulitis, abscess, gangrene, ulceration and slough may follow any infiltration or topical application of drug solutions used in regional block analgesia. Since all of these occur at the site of application, they are true local tissue reactions.

This chapter deals specifically with those *local toxic reactions involving primarily* the skin and mesenchymal tissues. Local toxic reactions involving the peripheral and central nervous systems as well as the vertebral column and the epidural (peridural) spaces will be found in Chapters 11, 23 and 24 on pages 112, 212, and 226 respectively. Drugs used in regional block not only include the anesthetic agents but also include drugs used as solvents and preservatives. These as well as *solutions used to sterilize ampules and instruments* may also cause local tissue reactions and therefore will be included in the discussion.

ETIOLOGY

Local tissue reactions of the skin and *mesenchymal tissues may be caused by (1)* faulty technique, (2) local anesthetic drugs, (3) vasoconstrictor drugs, (4) drugs used in regional block other than the local anesthetic agents and vasoconstrictor drug, and (5) mechanical pressure from indwelling spinal or epidural catheters.

Faulty Technique.—In this category are found those factors which depend upon the ability and conscientiousness of the physician *performing the block as well as upon the personnel* on whom he relies to clean, sterilize and prepare supplies necessary for the procedure.

Improper Sterilization and Cleaning of Instruments.—Equipment used to produce regional block analgesia should be washed carefully *with soap and water and rinsed adequately* with water, alcohol and ether, a technique familiar to most nurses. Haemo-Sol and other special detergent preparations, i.e., Detergex, etc., should not be employed without particular care—if their use is not followed by thorough rinsing, damage to tissues may result.[1,2] Dry sterilization by autoclaving using a sterilizer indicator (Propper Sterilizer Control Diack, etc.) is perhaps the most satisfactory method of assuring proper sterilization (see Chapter 22, page 206 for specific sterilizing instructions). Cold sterilization of needles, syringes and other *instruments in phenol or* formaldehyde solutions is dangerous unless the soaking is carefully controlled to assure adequate sterilizing and the equipment is conscientiously rinsed to remove all traces of the sterilizing solutions. Otherwise tissue damage may result either from infection or *from reaction to the sterilizing solution.*

Improper Preparation and Sterilization of the Drugs To Be Used.—All drugs used in regional block *procedures must be sterile*, otherwise infection, abscess and other complications are to be expected. With the exception of Xylocaine 2% without Adrenalin (20 cc. vial), which can be purchased from the pharmaceutical houses conveniently only in sterile solutions, other solutions used at the Mason Clinic for local infiltrations and peri-

pheral nerve block namely Pontocaine and Novocain are prepared from crystals which are received in sterile ampules Sterile commercially prepared saline solution (Abbott 100 cc size) is used as the solvent The author never uses hospital prepared solutions The ampules of the crystals of the local anesthetic drugs the solutions of Xylocaine the solvent and the 1 1000 Adrenalin solutions are *all* heat sterilized along with the needles and syringes in the regional block trays For the specific technique of autoclaving and a list of other drugs which may be autoclaved see Chapter 22 page 207 to page 208 At the present time cold sterilization of these drugs and equipment would probably not be considered as the best means of preventing contamination should a medico legal suit develop (see Chapter 23 page 221)

Wrong Solutions — Incorrectly identified solutions have been used with enough frequency even in the best of institutions that constant vigilance against such accidents can never be relaxed Using the wrong concentration of the drug or a solution that was thought to be one agent but in reality was another has been the usual "faux pas" Ether or alcohol has been injected for Novocain *and topical solutions which are usually* of high concentration have been used for local infiltrations and for peripheral nerve blocks The number of accounts of these incidences in the medical literature does not indicate the frequency of their occurrence since this type of accident is usually not reported

Contamination of the Anesthetic Solution by Solutions Used to Prepare the Skin Prior to Block —The medicine cup which is to contain the solution for preparing the skin prior to the block should be placed in front of the mixing graduate so that the solution used to prepare the skin will never pass over the anesthetic solution If the anesthetic solution is inadvertently contaminated and then used a slough of the tissue *or a neurological* complication may ensue

When the skin is aseptically prepared the *solution used* particularly if it is a tincture must either be allowed to dry or if there is an excess wiped off If this is not done then it is possible to introduce such a solution into the skin or subcutaneous tissue when making the injection The result may be inflammation of the tissue or slough

Incorrect Method of Administering the Agent —If solutions containing oil phenol alcohol propylene glycol or polyethylene glycol are placed too superficially or pooled (i e a large amount of the drug placed in one spot) necrosis of the tissue and sloughs are likely to occur The instructions which accompany these products usually specify *the fashion in* which the drug may be administered and caution against superficial injection and pooling[3 4 5] However Hackmeyer[6] believes that pooling is only a matter of degree and states " it seemed, in spite of the suggested technic [use of small gauge needle etc] that the oil must pool on injection that with different operators or the same individual at different times for a given volume of oil injected in whatever manner the only variants possible were the size of the pools and their number Pooling could be avoided relatively only "

Trauma —Excessively rough prodding of the tissues with needles by the physician per*forming a nerve block technique is to* be condemned since hemorrhage and damage to the tissue may result

Tissue Reactions to Local Anesthetic Drugs —*Swelling of the Tissues* —In dentistry persistent swelling of the tissue following the use of Xylocaine and Novocain occasionally appears in which there is usually no evidence of cellulitis hemorrhage or excess trauma "[1] The swelling is localized to the area of injection Lundquist *et al*[10] concluded from experimental work in animals that this swelling is due to the tissue irritation caused by copper nickel *and zinc ions released by these* two drugs from metal equipment such as receptacles syringes and needles They found *that tissue swelling is especially* pronounced following the injection of solutions containing copper but is comparatively insignificant after the injection of solutions containing nickel and zinc

Wiedling[11] contributed further to this investigation when he showed that this kind of tissue reaction did not occur unless Adrenalin is incorporated in the solution before the injection. He concluded that when the Adrenalin was omitted from the solution rapid absorption of these metallic ions occurred and they did not remain in contact with the tissue long enough to be irritating.

Several instances of this type of tissue swelling following the use of "carpules" of Pontocaine have also been called to the authors' attention.[12] Winthrop Stearns[13] attributed this complication to a tissue reaction to zinc oxide. In the manufacture of the synthetic rubber stoppers for these "carpules" an excessive amount of zinc oxide was used and the rubber stopper, on coming in contact with the Pontocaine solution, released a large quantity of zinc ions.[13]

When this type of persistent swelling occurs the physician or dentist need not become alarmed since it usually resolves in four to five days with no evidence of tissue damage or much pain to the patient.[7,9]

Inflammation, Sloughs and Ulceration.—Normal (isotonic) saline solutions of the common local anesthetic agents, i.e., Novocain, Pontocaine, Metycaine, Xylocaine and Intracaine, in the recommended concentrations do not usually per se cause tissue damage.[14-16] The minimal tissue changes, i.e., vasodilatation, edema and diapedesis of red blood cells reported by Meeker[17] and de Vincentiis[18] after intramuscular and subcutaneous injections of 1% Novocain hydrochloride combined with Adrenalin are insignificant and do not cause the patient difficulty.

However, if strong solutions of Novocain, i.e., 2% or greater *without* Adrenalin, are used to make a skin wheal which measures more than ¾ inch (2 cm.), a slough may occur.[1-] Hypertonic or hypotonic solutions of these local anesthetic drugs may also result in distention or shrinkage of the cells by exerting an osmotic pressure greater or less than that of the cell. This may cause either transitory or permanent damage to the tissues.

The use of Nupercaine too has sometimes caused local toxic tissue reactions.[16] It has caused no trouble of this nature when employed to produce spinal analgesia nor when used as a topical anesthetic. Nevertheless, in spite of its relatively common use in the above types of regional block techniques, its use in local infiltration, field block and nerve block procedures has been limited by reports that tissue sloughs have occurred following local infiltration. Brandesky[19] reports two such cases and Stohr[9] four.

On the other hand some authors believe that Nupercaine is a very satisfactory agent for local infiltration analgesia.[21-23] Keyes and McLellan[21,22] performed local infiltration anesthesia with a 1:1000 solution of Nupercaine for cystotomies, circumcisions, hydrocelectomies, excision of suprapubic fistulae, epididymectomies, and incisions of abscesses "with entire satisfaction." They have also used a 1:200 solution "repeatedly" for meatotomy and never encountered necrosis at the site of injection. Therefore they concluded that the drug is safe for infiltration and that the optimal concentration of Nupercaine for infiltration analgesia is a 1:1000 solution with a maximum of 100 cc. Bonica[23] also states that he is firmly convinced that Nupercaine properly used is an excellent local anesthetic affording prolonged analgesic blocks. These writers suggest that the skin necrosis following the use of Nupercaine would be more properly attributed to Adrenalin (epinephrine) or infection. The fact still remains that sloughs have occurred in conjunction with the use of this drug.[16,19,20]

Review of the means whereby the various local anesthetic agents produce anesthesia suggests the possibility that Nupercaine per se may be responsible for the slough. Most of the common local anesthetic agents, i.e., Novocain, Pontocaine, Intracaine and Metycaine, produce anesthesia by a selective reversible action on the nerve fibers or cells. *Reversible* ultramicroscopic coagulation of protein occurs within the cell. This reversible effect involves a number of physiochemical phenomena, the most important of which is a depolarizing of the nerve fibers or cells.[2,24]



Figure 21 Allergy to self administered Adrenalin for relief of asthma (contributed by Rowe and Rowe [?])

Depolarization occurs in the following fashion (1) The salt of the local anesthetic drug i e the hydrochloride is injected into the body (2) The salt is hydrolyzed by the small amount of alkali in the body (3) In this manner the base which is more soluble in the lipoid nerve structure than the salt is liberated (4) The ion of the anesthetic base carries a positive electric charge and this cation is readily attracted to the surface of negatively charged nerve structures As a result of this physiochemical process the permeability of the cell membrane is decreased the phasic shifts necessary to conduction of impulses prevented and the transmission of these impulses ceases or is slowed Thus we see that when Novocain Pontocaine Intracaine and Metycaine produce anesthesia they do so by a process which is *reversible* as the drug is detoxified

On the other hand Nupercaine and other drugs of the quinine type do not produce anesthesia of the nerve fibers and cells by this selective reversible action but by "irritation and partial or total destruction of the tissue including the nerve cells "[76] Therefore it is possible that Nupercaine per se may cause tissue damage and may result in a slough if placed in the epidermis or dermis during local infiltration

Cyclaine (hexylcaine hydrochloride) has been introduced recently as a safe local anesthetic agent But Sadove[81] reports four cases of slough following the use of 1% Cyclaine solutions intradermally to raise skin wheals The Cyclaine solution contained either 1 200 000 or 1 100 000 Adrenalin He believes that while Cyclaine seems to have a low systemic toxicity it has a high local toxicity when injected into the skin Recent work in animals by Mannheimer *et al*[9] showed that injection of Cyclaine into the cutaneous tissues and muscles resulted in infiltration of leukocytes and slough They observed no change in nerve tissue These animal experiments substantiate the clinical impression gained from injections in man that Cyclaine may on occasion cause a local tissue reaction

Tissue Reactions to Vasoconstrictor Drugs —The local tissue reactions considered under this heading are those which are believed due to prolonged anemia of the tissues brought about by the vasoconstrictor action of the drug

Acquired Allergy—A number of reports in the literature show that repeated small doses of Adrenalin (epinephrine) i e 0 2 cc to 0 5 cc of 1 1000 solution may sensitize a patient to subsequent use of the drug [33] To date most cases reported have occurred in asthmatics who have had to resort to self administration of Adrenalin for relief (see Figure 21 page 96) The reaction in these cases is due to allergy but in all probability the resulting lesion is referable to the tissue anoxia caused by a prolonged localized vasoconstriction This type of local toxic reaction is generally attributed to an Arthus phenomenon [34] Arthus showed that if horse serum is repeatedly injected subcutaneously in rabbits an anaphylactic shock reaction does not

occur but edema appears at the site of the later injections and this reaction may progress to abscess formation and even gangrene.

Prolonged Anemia of the Tissues from Persistent Vasoconstriction and/or Mechanical Pressure of the Injected Solution.—Persistent vasoconstriction which results in prolonged anemia of the tissues may be due to (1) too high a dosage of the vasoconstrictor drug for the patient's individual tolerance, (2) a lack of collateral blood supply to the area injected, e.g., finger or toe, (3) mechanical pressure from injecting too large a volume of the anesthetic solution, (4) any combination of 1, 2 and 3, or (5) constant continuous infusion of a vasoconstrictor drug to correct a persistent hypotension.

Untoward local tissue reactions at the site of application from vasoconstrictor drugs used in local anesthetic solutions are infrequent. Adrenalin has been reported responsible for some.[3, 4] Haller[7] in relating his experiences with the use of Adrenalin over an 18 year period reported two cases of skin sloughs in the area of the nose and two cases of intranasal mucous membrane sloughs from the use of small amounts of the drug, i.e., one drop of a 0.1% solution mixed with 2 or 3 cc. of a 0.5% solution of Novocain (procaine). In these cases pallor of the skin and the mucous membrane preceded the slough.

In addition to these instances, gangrene of the digits following the use of solutions containing Adrenalin for finger and toe blocks has been reported (see Figure 22, page 97, and Figure 23, page 98).[38, 39] Most of these authors cite the use of a tourniquet at the base of the digit as a possible contributing factor and note that hot moist dressings are also probably contraindicated postoperatively, since these increase metabolism of the tissue at a time when the blood supply cannot compensate for such an increase. Patients with peripheral vascular diseases may be particularly susceptible to such a complication.

Instances of slough in other regions of the body have occurred where local anesthetic solutions containing Adrenalin were used. Worgan[4] cites a case of slough of the skin of the penis which followed a block using a Novocain-Adrenalin solution at the base of the penis. This case required skin grafting. Eckenhoff[40] reported skin sloughs as the result of intradermal injection of local anesthetic



Figure 22. Slough of the thumb following the use of Novocain (procaine)-Adrenalin (epinephrine) solution for a digital block (contributed by Kaufman[38]).



Figure 21 Allergy to self administered Adrenalin for relief of asthma (contributed by Rowe and Rowe)

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ce of fluid subcutaneously. After clinical observations and examinations of the tissue grossly and microscopically, he concluded that Levophed produces marked constriction of the venules as well as the arterioles. This results in tissue anoxia and consequently in extravasation of tissue fluids into the area. This edema fluid is not absorbed because of the marked constriction of the venules and if circulation to the area cannot be re-established by therapeutic measures, then gangrene and ulcerations result. This study would appear to preclude the use of this drug as a vasoconstrictor agent in local anesthetic solutions.

Drugs Used in Regional Block Procedures Other Than Local Anesthetic Agents and Vasoconstrictor Drugs.—Many agents deposited in the body tissues may cause swelling, abscesses and sloughs. The following are those which have been commonly employed in regional block procedures as a blocking drug per se, as the solvent for the blocking agent, or as the preservative for the mixture and which have resulted in tissue damage.

Lytic Nonoily Drugs.—Alcohol, phenol, polyethylene glycol, propylene glycol and Efocaine are all neurolytic drugs and produce prolonged chemical blockade of the nerves by wallerian degeneration.[14, 3, 48, 60] They are not only capable of neurolysis but they also cause destruction of the surrounding skin and mesenchymal tissues resulting in swelling, cellulitis, abscess and slough.[14, 3] After the injection of the above mentioned lytic drugs into the muscles of animals and histological tissue studies of these muscles Mannheimer *et al*[14] and Nowill *et al*[3] found the following: (1) within a half to one hour after the intramuscular injection the area injected becomes pallid and a pale coagulated appearance is revealed on gross histological inspection; (2) in two days or so degeneration and necrosis of the muscle bundles is prominent with a marked infiltration of lymphocytes and macrophages; (3) between the 7th and 10th days multinucleated regenerating muscle cells, newly formed capillaries and fibroblasts indicative of an active regenerative process are noted; and (4) by the 12th to 21st days the necrotic debris is removed and regeneration is almost complete. All of these drugs reveal the same pathological changes in the muscle although the length of time each stage persists varies slightly with the drug used.[14, 3] While the muscle fibers may regenerate in this short a period of time (12 to 21 days), nerve fibers may take 6 to 9 months or longer to recover fully (see Chapter 11, page 116).

It should be emphasized that Efocaine, a buffered mixture of Novocain (procaine), polyethylene glycol, propylene glycol and preservatives, was *originally* thought to be innocuous and to produce long lasting analgesia by the precipitation of Novocain crystals and their slow absorption.[4, 3] This hypothesis was based on the fact that propylene glycol, while hygroscopic, does have a low systemic toxicity, and investigators studying it and polyethylene glycol, while noting some tissue damage following its injection into the mesenchymal tissues of animals, felt that it would be a safe vehicle for subcutaneous or intramuscular injection of drugs.[4, 60, 61] Carpenter and Shaffer[61] stated "Intramuscular injection of 5 to 10 times the expected human dosage level of PEG 300 or propylene glycol produced ischemic necrosis of the muscle fibers where the dose infiltrated a muscle bundle. The tissue response may be characterized as a mild chemical inflammation. This reaction is a transient one as no evidence of injury was found 14 days after the injection. Weinberg[4] concluded from experiments on rabbits: It is evident . . . that Efocaine *does not provoke a foreign body reaction* and does not remain in a demonstrable form in the tissues. The striking apparent resolution occurring in the injected muscles without a focal fibrous reaction remaining subsequent to the immediate necrosis, is further evidence of the lack of any permanent injurious action of the drug. The fibroblastic proliferation seen about the nerves during the early post injectional stages was found to disappear completely, *confirming the impression that one is dealing with a benign process which is apparently reversible* or which at least loses its char

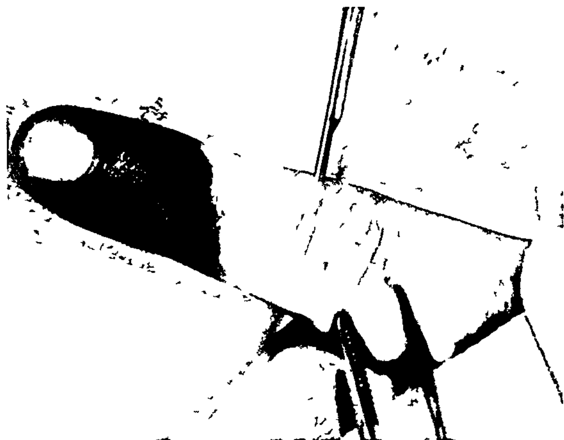


Figure 23 Gangrene of a finger following the use of a Novocain Adrenalin solution for a digital block (contributed by Adriani J)

solutions containing Adrenalin and advised against the use of vasoconstrictor drugs in local anesthetic solutions when making a skin wheal Serafin[41] reports tissue sloughs from local anesthetic solutions containing 1 20 000 Adrenalin and Kaufman[38] cites cases of slough of the abdominal wall and scrotum following the use of Novocain Adrenalin solutions It is debatable whether such local toxic reactions were caused by the vasoconstrictor drug or by the use of too large a volume of solution in areas where even small amounts of solution may create mechanical pressure adequate to embarrass the blood supply to that area, or by a combination of these two However since gangrene with the exception of that cited after skin whealing (page 95) has not been reported following blocks for which local anesthetic solutions without Adrenalin were used the use of vasoconstrictors must be considered as contributory

Untoward local tissue reactions following local infiltration of anesthetic solutions containing Neosynephrine and Cobefrin have not been noted Other new and powerful vasoconstrictor drugs i e Levophed (levarterenol bitartrate) Vasoxyl (methoxamine hydrochloride) Wyamine (N methylphenyltertiary butylamine sulfate) Oenethyl (2 methyl amino heptane hydrochloride) are being rapidly introduced on the market As yet they have not been widely used as substitutes for Adrenalin Cobefrin or Neosynephrine in local anesthetic solutions This is wise because following extravasation of the new and powerful vasoconstrictor agent Levophed tissue sloughs have been reported (see Figure 36 page 150 Figure 37 page 151 and Figure 38 page 152)[43 46] Therefore these drugs should not be incorporated in local anesthetic solutions until extensive animal research has been carried out to determine what if any is a safe concentration

Nyhus[47] seeking the cause of these sloughs reproduced them in dogs by injecting solutions containing 4 mg of Levophed per 1000

tion is always followed by regeneration. (4) Oil as a vehicle is somewhat of a nuisance. It is difficult to handle and is slowly absorbed from the tissues. Pooling of the oil is of secondary importance. Pooled oil presents the problem of a foreign body in the presence of inflammation caused mostly by the benzyl alcohol. This predisposes to abscess formation. (5) Secondary infection plays a part in production of abscess following injection of the oil soluble anesthetics. Lymphatic drainage into the injected area is considered of major importance in the production of septic abscesses. The precept that superficial injection predisposes to abscess formation is modified to take into consideration this factor; the site of the injection is also important. Superficial injections are more prone to result in abscess the farther away they are from the anal canal (except posterior midline) and in the path of lymphatic drainage.[7]

The use of oil preparations entails various disadvantages. Care to avoid superficial placement and pooling must be taken or sloughing of the tissues, cysts and foreign body granulomas may result.[1, 73, 71] Brown *et al*[1] showed that oils when injected intramuscularly caused inflammatory reactions and oil cyst formation varying from mild reactions with thin walled cysts and mild leukocytic infiltration to marked reactions with fibrinous thick walled cysts, edema, heavy leukocytic infiltration and muscle necrosis. Sesame oil caused the mild reaction and peanut oil the severe type. Furthermore, Truman[7] has reported that oil preparations injected into the operative site delay healing of tissue, increase inflammation and produce a larger area of scarring.

While Steinbrocker,[73] Bettman,[74] Wertheim and Rovenstine[7] and others have reported excellent results following the use of oil preparation, Hackmeyer,[75] Bonica,[3] Kelly[76] and the author have not been impressed with the uniformity of action of these drugs, particularly when injecting large nerves such as the intercostal nerves. Because of this and the tissue damage which may occur with these mixtures, these preparations are seldom used at the Mason Clinic.

Drugs Used to Potentiate Action of Local Anesthetic Agents.—Various agents have been used to potentiate the action of local anesthetic agents. These include organic bases, e.g., quinine derivatives; organic acids, e.g., hippuric acid; analgesic drugs, e.g., 3-acetyl salicylic acid (aspirin); proteins, e.g., egg albumin; and cations, e.g., potassium.[76] Most of these will cause local tissue irritation and seem to be of little value.[76] As a result they are not used by most physicians.

Ammonium Sulfate.—Mannheimer *et al*[77] have shown that concentrations of 5 to 7% ammonium sulfate do not cause degeneration or necrosis of the tissues. Recently Abbott has introduced for clinical experimental use a number of P.A.B. solutions. These contain procaine, ammonium sulfate and benzyl alcohol and differ only in that the concentrations of these three drugs vary in the different solutions. To date, tissue studies are not available. The maximum amounts of benzyl alcohol and ammonium sulfate used in them are 2.0 and 2.5% respectively. At the Mason Clinic the solutions have been employed in deep nerve blocks in cancer patients without noting either marked pain relief, improvement of the patient or significant pain at the site of injection. Nevertheless, pain and swelling of tissues on superficial placement of these mixtures have been called to the author's attention by Bonica.[7]

Hyaluronidase.—Hyaluronidase does not cause degeneration or necrosis of the tissues. However, local vascular phenomena, i.e., blanching and erythema, have been seen in the region infiltrated and a severe vaginitis of 24 hours' duration following a suprascapular nerve block has been observed after its injection.[78]

Diodrast (iodopyracet).—Diodrast has been used in regional block analgesia to visualize the spread of anesthetic solutions. To date sloughs and abscesses following its injection have not been noted. On the other hand, the author and his residents have injected Diodrast subcutaneously in themselves and have found that it creates an ecchymotic area at the site of injection which remained painful for two to five days. And following deep place-

acteristics and is absorbed into the surrounding tissues. With full realization that injection of 0.5 cc of Efocaine into a nerve bundle as small as the brachial plexus in the rabbit is an enormous dose, it is not surprising to find the presence of focal degeneration of the myelin sheath. The mechanical effects of injecting 0.5 cc of any substance into such a small confined space are to be considered of paramount importance in interpreting the histologic features found. Apparently the fibroblastic proliferation and bland thrombus formation found in the sections of the lip are of the same benign character as those found in the other tissues since *no histopathology was evident in the sections of those animals followed for a long period of time*."

To rule out the possibility of tissue reactions being *mechanical* in origin, isotonic sodium chloride solutions have been injected under identical conditions to serve as a control. No degeneration or necrosis of the muscle bundles was noted.[14] Further studies contradicting Weinberg's conclusions have shown fairly conclusively that the long analgesia produced by Efocaine is a result of tissue destruction, particularly of the nerves and the nerve end plates—not slow absorption of the Novocain crystals.[14, 15, 61, 3] The prolonged anesthetic action of Duracaine and Diothane hydrochloride was found by Maykut and Ryan[65] to be similarly caused by tissue damage.

Oil Preparations With or Without Benzyl Alcohol as a Preservative.—Oil bases have been used to slow the absorption of the anesthetic agent since Benacol (Novocain base dissolved in oil) was first introduced in 1927 by Yeomans, Gorsch and Mathesheimer.[66] These authors believed that the oil released the local anesthetic agent slowly, producing prolonged analgesia. This hypothesis was accepted for a number of years until it was shown that when these solutions were used degeneration of the tissue occurred.[67, 68] It is interesting to note that there were three incidences of slough in the 25 cases reported by Yeomans *et al*.[66]

Duncan[69] working with cats found that when oil solutions containing benzyl alcohol were injected subcutaneously around the facial nerve, degeneration of that nerve occurred in every case. Duncan and Jarvis[69] also point out that when the oil solutions contained benzyl alcohol in concentrations of 5% or greater, the benzyl alcohol per se was responsible for most of the tissue destruction. They state: "It is concluded that the prolonged effects of the anesthetic mixtures in oil were caused almost entirely by the benzyl alcohol content, and this substance in 10% concentration will destroy all of the fibers in small nerves and a considerable number of them when used in 5% concentration." However, weaker concentrations of benzyl alcohol, e.g., 0.75% or less used as a preservative in Dolamin, an ammonium sulfate solution, causes no degeneration or necrosis of the tissue.[14] Therefore it is likely that whether or not benzyl alcohol exerts a deleterious effect on the tissue would depend on its concentration.

Since most oil preparations contain benzyl alcohol, it may be concluded that the problems encountered with these mixtures are caused by both the benzyl alcohol and the oil, benzyl alcohol probably being the greater offender. The local anesthetic drug incorporated in these mixtures seems to play little part in either the prolonged analgesia or the tissue destruction. Hackmeyer[6] in his summary makes the following pertinent points on this subject: (1) Benzyl alcohol is a tissue irritant that is capable of causing degeneration of human muscle, of destroying nerves and of causing sufficient tissue reaction at times to produce an aseptic abscess. (2) Benzyl alcohol seems to play the major role in producing prolonged anesthesia or hypesthesia, probably by chemical degeneration. Larger doses produce complete anesthesia, smaller doses hypesthesia. Other drugs are relatively unimportant for the production of these effects. (3) The effect of benzyl alcohol is controlled better in solutions of oil than in water, the oil allowing higher concentrations, slower diffusion and prolonged action with less resulting irritation and more effective denervation. It is noted that nerve degenera

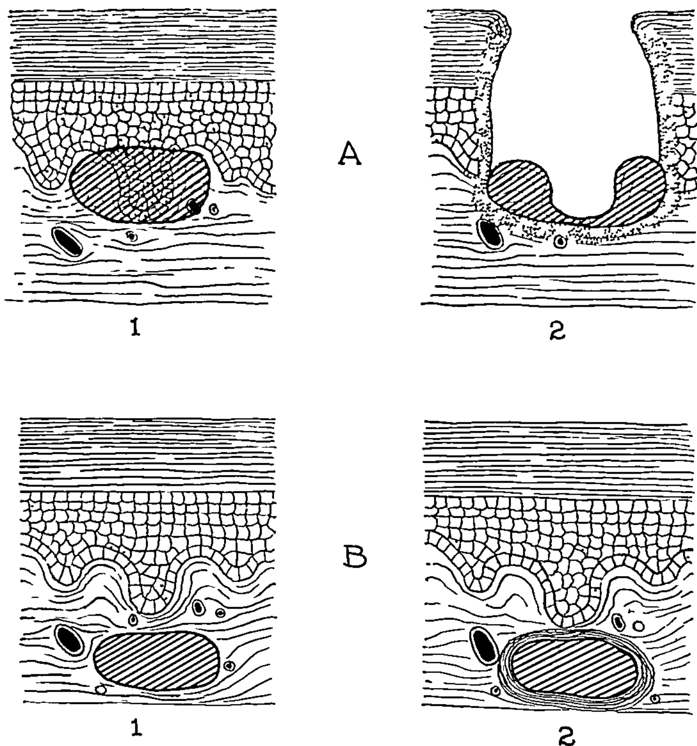


Figure 24 Mechanism of slough (A) Neurolytic solution placed immediately under the skin (1) damages tissues destroys the blood supply to the skin immediately above sterile abscess and results in slough (2) (B) Neurolytic solution placed deep in tissues (1) produces same damage but since the blood supply on none of the sides of the sterile abscess is damaged no slough results and the phagocytes in the tissues exert their scavenger effect The damaged tissues regenerate or the tissue scars (2)

ment of this drug as in stellate ganglion block pain which lasted two to five days has been reported to us by approximately 20% of the patients into whom it was injected[79]

Pyribenzamine (tripelennamine hydrochloride)—The anesthetic properties of this drug administered topically have been demonstrated[98, 104] Recently it has been used by Betcher *et al*[105] for peripheral nerve blocking in clinical experiments They comment Pyribenzamine was used for local infiltration and regional nerve block for diagnostic therapeutic and surgical procedures in 459 cases All injections resulted in a rapid onset of action longer duration of action a higher percentage of success in regional nerve blocking and a smaller number of systemic reactions [than Novocain] There were indications of some burning occasional bleeding erythema at the site of injection and possibly increased oozing which may be owing to increased vascularity or spreading ability of the drug or both Complete recovery was noted in all patients" They also state It is realized however that the effects of Pyribenzamine injected intraneurally and perineurally in experimental amounts should be studied histologically as was done with long acting local anesthetics by Mannheimer, Pizzolato and Adriani"[14]

Indwelling Spinal or Epidural Catheters—Reports of catheters causing irritation and infection at the site of entrance into the body have appeared[80] This type of reaction might reasonably be expected but it occurs very infrequently

SIGNS AND SYMPTOMS

The signs and symptoms of a tissue reaction following a local toxic reaction to the drugs used in a regional block are primarily those of (1) burning (2) swelling (3) inflammation (4) ulceration and gangrene with damage to the peripheral nerves or their end plates and (5) a combination of one or more of these

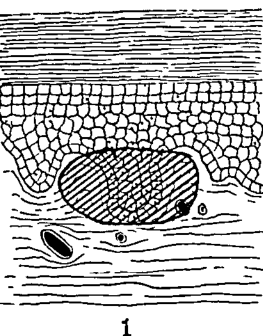
Burning—This symptom may occur following the injection of local anesthetic solutions in which the solvent is distilled water It has also followed the injection of Cyclaine (hexylcaine), Efocaine and alcohol Burning on injection may signal a more serious complication

Swelling of the Tissues—This is usually expected during superficial infiltrations with local anesthetic solutions It should cause no problem except perhaps in plastic surgery where it may be reduced by the use of hyaluronidase as will be noted later In most instances it is due to the injection of such an excessive amount of the local anesthetic solution that the tissues cannot accommodate it but on occasion the inherent property of the area injected may be responsible In the occasional case swelling may signify a reaction to metallic ions it may also signify an abscess slough and ulceration a serious hemorrhage (see Chapter 9 page 89) or a subcutaneous emphysema from a pneumothorax (see Chapter 6 page 62)

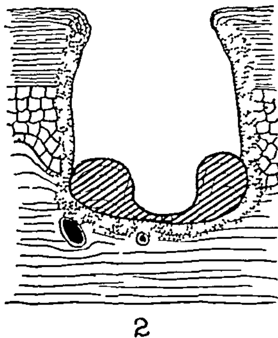
Inflammation—The chief complaint of a patient developing an inflammation (sterile abscess or cellulitis) is pain in the area involved and the well known signs are present redness local heat swelling and tenderness of the tissue involved

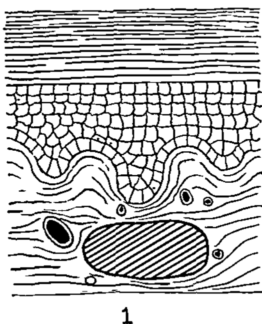
Ulceration and Gangrene—These are in most instances a result of tissue destruction from (1) drugs which are known to have a lytic effect on the tissues (2) an allergy to the vasoconstrictor drug or (3) prolonged anemia of the tissues from either a direct action of the vasoconstrictor drug on the blood vessels of the area or a combination of this and mechanical compression of the vessels from injecting too much solution Infection is not necessarily a cause of ulceration and gangrene but secondary infection of a sloughed area may occur if proper therapy is not instituted

Local Signs and Symptoms of Ulceration and Gangrene—These vary depending on whether the lesion is superficial or deep The local signs and symptoms of a *superficial* lesion progressing to gangrene occur because all the terminal blood supply to that area is temporarily destroyed (see Figure 24 page 103) The signs and symptoms of ulceration



A





B

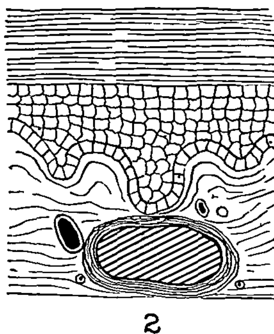


Figure 24 Mechanism of slough (A) Neurolytic solution placed immediately under the skin (1) damages tissues destroys the blood supply to the skin immediately above sterile abscess and results in slough (2) (B) Neurolytic solution placed deep in tissues (1) produces same damage but since the blood supply on none of the sides of the sterile abscess is damaged no slough results and the phagocytes in the tissues exert their scavenger effect The damaged tissues regenerate or the tissue scars (2)

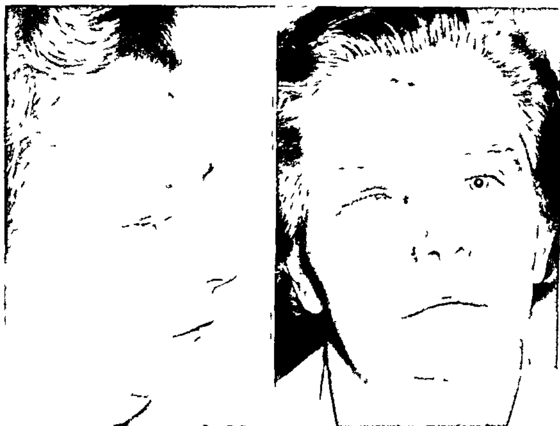


Figure 25 Swelling of the face following the injection of 1 cc of absolute alcohol into the infraorbital branch of the maxillary nerve both at the infraorbital foramina and in the infra orbital canal for trigeminal neuralgia There is little doubt that the alcohol caused degeneration of the tissue but that deep placement precluded a slough As is usual this swelling resolved in 7 days with no specific treatment

and gangrene are well known and are as follows (1) paleness and cadaveric appearance of the area *injected are prominent initially* (2) tenderness and pain occur immediately following injection unless the anesthetic property of the drug is sufficient to mask them (3) swelling may occur (4) the skin becomes a dusky color (5) a small purplish patch or patches with ill defined margins appear (6) at the same time a large bulla or blister having a yellowish appearance may erupt over the dusky area or somewhere else on the red skin and (7) the necrotic area may slough, leaving a raw denuded area A foul smell usually accompanies wet gangrene depending on the size of slough No odor accompanies dry gangrene such as that which occurs if the blood supply to a digit is compromised

On the other hand when the lesion is *deep* in the tissues superficial skin and subcutaneous tissue sloughs seldom occur although necroses of the deep mesenchymal tissues and *nerves in the region of the deposited solution* do result see Figure 25 page 104 and Figure 26 page 105[14 1] The local signs and symptoms of *deep* tissue necrosis and degeneration are often confined to swelling and this usually occurs within 1 to 2 hours after injection Tenderness and pain may become noticeable as the anesthetic qualities of the injected drug are dissipated The blood supply around the devitalized area is sufficient to combat this kind of problem and healing occurs either by tissue regeneration or scarring (see Figure 24 page 103) The mesenchymal tissue will usually heal in two weeks to a month but it may take a peripheral nerve nine months to a year to regenerate (see Chapter 11 page 116)

Systemic Signs and Symptoms of Ulceration and Gangrene—Since large superficial areas

of gangrene and ulceration seldom follow regional block, systemic signs and symptoms, i.e., increased body temperature, malaise, etc., from absorption of toxic products occur infrequently. The tissue degeneration which occurs following the *deep* placement of long-acting solutions seldom is complicated by infection. It is, so to speak, a "sterile abscess."

When systemic signs and symptoms do occur, they are usually due to secondary infection of a superficial sloughing area which has not been properly treated.

PROPHYLAXIS

Do Not Use Excessive Volumes of Solution.—The use of the minimum amounts of local anesthetic solutions necessary for the establishment of a successful block and strict adherence to the block techniques will usually prevent undue swelling initially and help to prevent pressure necrosis from developing later.

Avoid the Use of the Wrong Solutions.—To avoid using wrong solutions, the physician should carefully check all stock solution. If there is any doubt about the solution, it *should be discarded*.

Avoid Breach in Technique and Possible Infection.—Inflammation, abscess, cellulitis, sloughs and even gangrene may result from (1) the use of unsterile equipment, (2) contaminated drugs, (3) blocking of a patient with a septicemia, or (4) introducing a needle through an infected area.

To avoid infection and its complications the instruments and drugs must be adequately sterilized (see Chapter 22, pages 206 and 207, for technique). In addition the physician must use an aseptic technique during the block procedure, i.e., the same sterile precautions should be taken as for an operation. These include (1) sterile precautions when opening the block tray, (2) examining the sterilizer control, (3) wearing gloves and (4) *correctly preparing the area to be injected*.

The doctor should wear sterile gloves for at least two reasons: (1) they are no more hindrance to the doctor performing a block than to a surgeon performing a delicate operation and they do offer an additional safeguard to the aseptic technique, and (2) when a doctor executes a large number of regional procedures, wearing gloves is advisable for his own protection, as occupational sensitivities to local anesthetic agents are not uncommon (see Chapter 14, page 127).

The patient's skin should be prepared with one of the routine surgical preparations. If the patient is to leave the office or hospital following the block and cosmetic appearance is a consideration, a 5% iodine solution washed off with alcohol or aqueous (colorless) merthiolate is satisfactory. The block field should be carefully walled off with sterile towels to assure asepsis.

Trauma during the placement of the needles must be avoided, as devitalized tissue and aggregates of blood are excellent culture media for bacteria.



Figure 26. Swelling of the face following a block with absolute alcohol of the maxillary nerve as it leaves the foramen rotundum to cross the pterygopalatine fossa. There is little doubt that the alcohol caused degeneration of the tissue but that deep placement precluded slough. The swelling receded within one week.

Avoid Lytic Drugs—Drugs known to cause severe tissue damage should be employed only to control intractable pain and in these cases the patient and his family should be made to understand the complications which may result Signed permissions are a safeguard against legal action (see Chapter 12 page 119) Blocking with these drugs should be left to the expert

Although Nupercaine solutions have been used for local infiltration and nerve blocks by a number of physicians and in many cases without causing local tissue reactions it must be remembered that necroses and sloughs following the injection of the drug in these techniques have been reported Therefore it is the author's opinion that the use of Nupercaine should be restricted to spinal and topical administration and not used for infiltration because other agents particularly Pontocaine will produce analgesia of approximately the same duration by a selective reversible action on the nerve fibers and cells and will not damage the tissues" [6, 8, 87]

Careful Selection and Use of Vasoconstrictor Drugs—While local tissue damage from vasoconstrictor agents at the site of administration is rare during regional block procedures it has been reported This renders it obvious that before using a vasoconstrictor drug in a local anesthetic solution the physician should carefully evaluate the indications for its use the patient's physical condition the site to be injected and the vasoconstrictor drug to be employed If the following precautions are carefully adhered to local tissue damage from vasoconstrictor drugs due to an acquired allergy mechanical compression of the blood vessels and prolonged vasoconstriction with tissue anoxia will be minimized

Use Standard Vasoconstrictor Agents—The time-tested vasoconstrictor drugs which have been in clinical use for many years are Adrenalin Cobefrin and Neosynephrine They seldom cause degeneration and necrosis of the tissues when used in local anesthetic solutions in the recommended dosages The newer agents which exert a strong vasoconstrictor effect on the blood vessels but do not affect the heart or cardiac output do not enjoy the reputation of having stood the test of time and one Levophed is known under certain circumstances to have caused slough (see Chapter 15 page 150) Therefore, before using these agents promiscuously in regional block solutions physicians should be aware that they may cause untoward reactions

At the present time it is the author's opinion that the new powerful vasoconstrictor agents should not be used in local anesthetic solutions as a substitute for Adrenalin etc in hopes of prolonging the duration of the block Since Pontocaine Adrenalin solutions give a duration of action of four to six hours without causing tissue damage or a high incidence of systemic toxic reactions it would seem that the risk involved in substituting Levophed or one of the other new vasoconstrictor agents for Adrenalin is not justifiable until the safety of these drugs is proved experimentally in animals and humans

Avoid Vasoconstrictor Drugs when Blocking the Digits the Nose and the Penis—In these areas the volume of solution which may be accommodated without causing mechanical pressure on the blood vessels is small If perchance an excess amount of the local anesthetic solution containing a vasoconstrictor drug is injected not only is there constriction of the vessels by mechanical pressure but also by drug action Therefore the chance of completely occluding the blood supply to the area is enhanced as evidenced by reports of this in the literature [3, 4]

The above cited physicians and a number of others have cautioned against the use of vasoconstrictor drugs when doing a digital block [3, 4, 88, 90] However Pitkin [91] who used Adrenalin and who had vast experience with local and regional blocking had not seen gangrene of the finger or toes from digital blocks and believed that the cases reported in the literature occurred because Adrenalin was employed in very high concentrations

It is the author's opinion that if a vasoconstrictor agent is used in the local anesthetic solution when performing digital block a block around the nose or a block of the nerves

to the penis 75 to 150 T R U (turbidity reducing units) of hyaluronidase should be incorporated in the solution to effect its wider spread and thus promote absorption of the solution and a consequent decrease of mechanical pressure (see Figure 2 page 15)[92]

Careful Examination of Patient Prior to Blocking.—Inasmuch as it has been shown that the occasional asthmatic patient may become sensitized to Adrenalin such patients must be carefully examined prior to injections of a large volume of local anesthetic solutions containing Adrenalin otherwise a large slough may occur If such a patient is found to have areas of scarring where self administration of the Adrenalin has taken place then no Adrenalin should be used in the local anesthetic solutions or a general anesthetic should be administered If it is imperative to use Adrenalin in these patients synthetic Adrenalin preparations should be used for it has been found that these are safe and do not result in the Arthus phenomenon i e necrosis and scarring[29]

Do Not Add Vasoconstrictor Agents to Local Anesthetic Solutions with Vasoconstrictor Action.—Solutions of cocaine are known to have a powerful vasoconstrictive action and if vasoconstrictor agents are added to these solutions this type of action on the blood vessels is greatly increased The result may be a slough

TREATMENT

The extent of treatment of local toxic tissue reactions depends on its type

Swelling.—In the greater number of instances tissue swelling from the injection of excessive amounts of the local anesthetic solution may be expected and is of little significance because it is self limiting and will regress without specific treatment However if the physician wishes to minimize tissue swelling he should incorporate 150 T R U (turbidity reducing units) of hyaluronidase (Diffusin Alidase or Wydase) in the local anesthetic solution and use gentle massage following the infiltration[92, 93] Hyaluronidase added to a local anesthetic solution markedly shortens the duration of anesthesia unless Adrenalin is also added

In addition to the tissue swelling caused by mechanical pressure persistent swelling of the face following block of the mandibular and maxillary nerves or their terminal branches may apparently have other causes Whether persistent swelling is due to a local allergy to the drug or the release of metallic ions and consequent direct tissue irritation is open to question Although innocuous such swelling does present a problem cosmetically[79] Since some dentists feel that it is a direct allergic phenomenon they advocate the use of the antihistaminic drugs prophylactically prior to injections and others use such drugs only as treatment after swelling has occurred[79]

It must be stressed that while tissue swelling at the site of injection is usually of little concern it may herald a *serious complication* i e infection slough, hemorrhage because of an unexpectedly prolonged clotting time or a pneumothorax Therefore swelling cannot be viewed lightly until a definite diagnosis is established The problems entailed by pneumothorax and increased clotting time are considered elsewhere (see Chapter 6 page 63 and Chapter 9 page 90)

Inflammation.—The active treatment of inflammation (abscesses and cellulitis) is well known and needs little discussion It includes hot wet packs and in some instances incision and drainage If secondary infection occurs antibiotic drugs may be necessary If the inflamed area ulcerates or sloughs and will not heal then debridement grafting etc as indicated below must be considered

Blebs, Ulceration and Gangrene.—When a compromised blood supply threatens the viability of an area of the body the area involved becomes blanched and appears cadaveric Immediate treatment to decrease the temperature of that portion of the body should be undertaken in order to lower the tissue metabolism An attempt to increase the circulation to the part by sympathetic blocking and/or intravenous Novocain infusions should be made and the part must be guarded from trauma Often if these steps are instituted

without delay progression to a more severe lesion may be stopped

When superficial blebs and vesicles appear the situation is more serious However if in addition to the therapy suggested above mecholyl iontophoresis is instituted the lesion may regress rather than progress This was true of one of the cases observed at the Mason Clinic (see Figure 39 page 152)

Small ulcerations and gangrene (0 5 to 1 0 cm) such as those produced by an acquired allergy i e the Arthus phenomenon or those occurring following the raising of an intra dermal wheal require no treatment other than keeping the lesion clean In most instances they heal in four to six weeks

On the other hand when large areas of ulceration result such as those following the superficial placement of lytic drugs a digital block or the administration of powerful vaso constrictors (Levophed) apppropriate medi cal and surgical treatment including antibi otics debridement and grafting must be in stituted Antibiotic therapy may be necessary to correct or prevent secondary infection of the sloughed area In instances where ulcer ated areas remain infected in spite of anti biotic treatment and cannot be prepared for grafting temporary *sympathetic denervation* by blocking the appropriate ganglion or gan glia may be indicated to increase the blood supply to the area[79] We have used this therapy prior to grafting wounds with marked success in cases where infection of the wound with pyocyaneus bacteria following surgery or trauma prevented the grafting of large areas The block apparently through in creased blood supply results in a reduction of the bacteria count of the wound and lessens the drainage making the area more fit for grafting

McKenzie[94] reports one case of slough of the corner of the nose after surgical section of the maxillary nerve for trigeminal neu ralgia The sloughed area could not be grafted until after the stellate ganglion was removed (see Chapter 12 page 122) This substantiates the efficacy of sympathetic block for preparing a graft site

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CHAPTER 11

Lesions of the Peripheral Nerves

THE TERM *"peripheral nerves" as used in* this chapter refers to those portions of nerves which extend beyond their point of emergence *from the foramina of either the vertebrae* or the skull

ETIOLOGY

When a lesion of one or more peripheral *nerves occurs following a regional* block *procedure* all too frequently the nerve block itself is criticized for producing the resulting *neuritis paresis or paralysis Since the literature* contains reports of neurological involvement of the nerves of the upper and lower *extremities following both regional block* and general anesthesia it does not seem justifiable to condemn a regional block technique automatically without a careful investigation of all the factors which might result in this type of complication[1,17,3] The following are fac*tors which have been thought responsible for* the peripheral nerve lesions which may occur after regional block procedures

Faulty Positioning of the Patient—Faulty positioning of the patient may occur either during the time of the operation or in the immediate postanesthetic period while the regional block is still effective Often patients receiving a regional block are heavily sedated either prior to the block or following its execution Consequently superficially placed or deeply placed nerves in unanesthetized or anesthetized areas may tolerate abnormal amounts of pressure or stretching without complaint on the part of the patient during the surgical procedure or the immediate postoperative period Paralysis of the ulnar nerve *at the elbow from faulty positioning of an arm* while anesthetized by brachial plexus block has been seen by the author on four occasions[13] Temporary paralysis of both lateral femoral cutaneous nerves has also been seen following spinal anesthesia and this case too was not due to the anesthetic but to pressure on these nerves while the patient was in the prone position undergoing a laminectomy for a ruptured nucleus pulposus It is obvious that in both cases the paresis was not the result of the block These paralyses cleared within three months

In addition to faulty positioning during surgery or the postoperative period trauma to a joint or a nerve of an anesthetized extremity may occur as a result of mishandling If an extremity which is blocked should fall off the operating table or slip out of the surgeon's hand and drop over the edge of the table either a nerve injury may result or a dislocation of a joint occur *This is particularly* true if the arm is not carefully protected after a brachial plexus block After this par*ticular block should the arm fall off the cart* because of a movement of the patient or should the physician accidentally drop it dislocation *of the shoulder is likely to occur*

Neurolytic Agents—Following the injection of these drugs overflow to adjacent nerves not intended to be involved may result in neuritis or paralysis The possibility of such an occurrence should always be anticipated whenever a neurolytic drug is to be injected and it should not surprise the physician who is willing to undertake the use of these agents (see Chapter 12 page 119)

On the other hand all too frequently the

physician may not realize that the drug he is injecting is neurolytic. *Therefore it is vital to keep in mind that alcohol, phenol, various oils (peanut, etc.), propylene and polyethylene glycol, excessive concentrations of local anesthetic agents, ether, formaldehyde* and *detergents* are all neurolytic drugs.[14, 7] Often the trade name of a preparation does not reveal its contents, e.g., Bromsalizol, which contains 4% mono-bromhydroxy benzyl alcohol in peanut oil and which is a neurolytic preparation.[26, 3] And because this is true, a physician must know the contents of the preparation rather than trust the advertising literature which terms it "safe." Furthermore, if ampules of drugs are placed in sterilizing solutions containing neurolytic drugs or if neurolytic agents are used to cold sterilize equipment to be used in local infiltration and peripheral nerve block, these neurolytic agents may in an insidious way gain entrance into the body.

Surgical Trauma.—During surgery, a cut nerve, a misplaced retractor with pressure on a nerve, edema around the nerve from handling the tissue and/or a reflex vasospasm of the blood vessels supplying the nerve may all result in paralysis or paresis of peripheral nerves.

Improperly Applied Casts.—Casts that are too tightly applied or not correctly padded may cause undue nerve pressure or compression of blood vessels with resulting neurological sequelae.

Improperly Applied Tourniquets.—A tourniquet applied to the upper or lower extremity must be correctly applied. When a tourniquet is applied low on the thigh or arm or if it is applied below the knee or elbow where there is little tissue padding such as muscle or subcutaneous fat and where bones are superficial, not only are the blood vessels constricted but damaging pressure is transmitted directly to the nerves. Moldaver[23] reports seven such cases which he describes as "Tourniquet Paralysis Syndrome." He emphasizes the following facts concerning this syndrome: (1) the syndrome is due to mechanical pressure on the nerve and is not the result of ischemia, (2) there is hypotonia or atonia of the muscles but no appreciable atrophy, (3) touch, pressure, vibration and position senses are usually absent, but pain sensation is never lost and heat and cold sensations are usually not affected, (4) there is no sign of neuroma at the site of injury and no Tinel's sign* present, (5) sympathetic fibers are not affected, (6) there is a block of conduction as shown by electrical studies above the injury, but good reaction below the injury, and (7) the level of the lesion may be determined by electrical stimulation.

In addition to this type of paralysis from direct pressure on the nerves, tourniquets which are allowed to remain inflated for times exceeding 1½ to 2 hours may cause nerve damage as a result of ischemia.

Neurolytic Action of Local Anesthetic Agents.—As noted previously, local anesthetic agents used in excessively concentrated solutions (usually 5 to 10 times that recommended) may have a neurolytic action. Lundy *et al*[16] demonstrated a 17% concentration of Novocain to have such an effect. However, local anesthetics used in the usual recommended concentrations do not cause damage to the peripheral nerves when injected perineurally, i.e., around the nerve.

To date my associates and I have seen one case of prolonged numbness attributable to the anesthetic agent following a peripheral nerve block. It occurred following a sciatic and femoral block procedure in which 50 cc of 0.25% Pontocaine solution had been used. The anesthesia time with Pontocaine solutions is usually 6 to 7 hours but in this instance it lasted for 72 hours, after which the numbness cleared and the patient had no further complaint.

Intraneural Injections.—Intraneural injections have been condemned as causing nerve damage by pressure or by a combination of this and action of the drug on the nerve tissue. This is open to debate. My associates and I have made what we believed to be intraneural injections into the divisions of the

* Tingling sensation distally when percussing over site of a nerve.

brachial plexus as well as into the sciatic nerve using 10 cc of solution without causing nerve damage Our criteria for intraneural injection are paresthesias and aching in the extremity during the injection of the anesthetic solution

Damage from Direct Contact of the Needle Point with the Nerve—This again is a debatable cause and my associates and I believe that it is seldom if ever responsible for peripheral nerve lesions In regional block procedures such as sciatic block, block of the brachial plexus or its branches mandibular block maxillary block etc our dictum at the Mason Clinic has always been "no paresthesias, no anesthesia"[24] To elicit paresthesias it is obvious that the needle must come into direct contact with the nerves and at times pierce the epineurium and enter the fasciculi yet to date we have seen no neuritis or other nerve lesions when aqueous solutions were used

Contamination of Solutions or Equipment—If infection is introduced into the nervous tissue or in close proximity to it and an abscess develops damage to the nerve may occur Introducing a filterable virus into the body is another possibility It has been noted that herpes zoster may follow a block of the gasserian ganglion

Pre-existing Lesions—Organic diseases such as metastatic carcinoma hemangioma etc may coincidentally exist in the region where a peripheral nerve block has been performed Following the block the normal course of the disease may be such as to exert pressure on a nerve When this occurs the patient may blame the block procedure for the nerve involvement

Likewise, injuries of the extremities for which surgery is being performed have often severed the nerves and blood vessels and these injuries should be noted in the patient's chart and pointed out to him prior to surgery particularly when block procedures are to be used so that temporary or permanent postoperative sensory and motor changes are not attributed to the block

Metallic Ions Released from Receptacles and Syringes—All of the published reports on the use of Xylocaine (lidocaine) hydrochloride have shown it to be a safe drug and one that does not cause irritation to the nerve Nevertheless, in the past year 19 cases of neuritis following brachial plexus blocks in which 1 or 2% aqueous solution of Xylocaine was employed have been called to the author's attention[26, 27, 28, 33] In reply to inquiry for a possible explanation the Astra Pharmaceutical Company cited the work of Lundquist *et al*[23] and Wiedling[30] Lundquist *et al*[23] showed that (1) under certain circumstances solutions of Xylocaine and Novocain (procaine) may liberate small amounts of copper nickel and zinc from hypodermic syringes made entirely or partly of metal and (2) that while any one of these metallic ions may cause tissue irritation and swelling the copper ions are usually the greatest offenders Wiedling[30] showed that if Adrenalin is not added to the solution these metal ions are absorbed before a local toxic reaction has had time to develop but that Adrenalin permits their contact with the tissue long enough to cause irritation

Recently two cases of brachial neuritis following brachial plexus block with Cyclaine (hexylcaine) have been noted and they may perhaps have been caused by this type of reaction[26]

Repetition of a Specific Nerve Block Within Twenty four Hours—It is not unusual for the question to be raised as to whether or not the repetition of a block procedure within 24 hours may result in nerve damage On numerous occasions my associates and I have repeated nerve blocks within 24 hours in which paresthesias had to be elicited to assure a successful block yet to date we have not seen one case in which neuritis resulted

SIGNS AND SYMPTOMS

When the anesthetic dissipates itself the patient immediately will complain of *motor weakness* or *paralysis pain*, and *impairment of sensation* i e tingling and numbness over *the area of distribution of nerves involved* These signs and symptoms of nerve involve

ment may last for one to three days and then disappear completely or they may persist for three to six months or longer

PROPHYLAXIS

To prevent lesions of the peripheral nerves

Avoid Pressure on the Nerves.—When placing the patient on the operating room table when applying a cast or when positioning the patient in bed after a regional block procedure every effort must be made to avoid pressure on the nerves, particularly those which are superficially placed and vulnerable to pressure The nerves most vulnerable to pressure include (1) the brachial plexus (2) the ulnar nerve at the elbow (3) the radial nerve at the wrist (4) the lateral femoral cutaneous nerve at the anterior superior spine of the ilium and (5) the peroneal nerve just below the knee

It follows that tourniquets as well as retractors must be carefully placed during surgery to avoid a damaging degree of pressure on the nerves and blood vessels It must be remembered that if the blood vessels are traumatized, vasospasm may develop which may simulate a peripheral nerve lesion and which, if persistent may actually result in secondary damage to the nerves Tourniquets should be deflated after 1½ hours A few minutes should be allowed for the blood to perfuse the arm adequately and oxygenate its tissue before the blood is again drained from the arm and the tourniquet reinflated

Avoid the Use of Neurolytic Drugs—Neurolytic drugs should be reserved for therapeutic blocks in patients with intractable pain from an incurable disease and in whom root section or cordotomy is not possible Even then they should be employed only if the patient is fully cognizant of the complications which may result from their use

The physician should either mix his own drugs or take other precautionary means such as tasting the solution before injection to assure himself that he has the correct drug All too frequently physicians have injected a drug prepared by the hospital pharmacy or handed him by the scrub nurse only to find that it was either the wrong concentration of the local anesthetic drug or the wrong drug (i e , alcohol or ether)

Extreme care should also be taken when handling solutions used to prepare the skin prior to insertion of the needle Many of these solutions, particularly those containing alcohol are neurolytic If they are inadvertently dropped into the local anesthetic solution or on the needles and go unnoticed they may come in contact with nerve tissue and cause trauma

Avoid Intraneural Injections.—Although it seems questionable that intraneural injections result in nerve lesions, it is perhaps best to avoid such injections since satisfactory anesthesia for surgery and diagnostic and therapeutic blocks may be obtained by injecting the anesthetic agent in close proximity to the nerve This does not mean that paresthesias should not be sought in those blocks in which they are indicated but merely that the needle should be slightly withdrawn if the patient complains of paresthesia during the injection of the drug It must also be remembered that if intraneural injections are made at or close to the foramina of the vertebrae or the skull that the drug may reach the spinal cord or brain via the perineural spaces (see Chapter 5 page 49)[31, 32, 33]

Do Not Use Excessive Concentrations of the Local Anesthetic Agent.—As pointed out previously the usual recommended dosage of a local anesthetic agent should not be exceeded and it is always best to use the weakest concentration of the drug which will accomplish the task at hand Excessively high concentrations of these drugs have been experimentally shown to be neurolytic[15, 16]

Use Sterile Precautions.—All the drugs and equipment to be used must be adequately sterilized to avoid the introduction of pathogens with consequent inflammation Although all solutions and drugs used in peripheral nerve blocking and local infiltration by the Anesthesia Department of the Mason Clinic are purchased as presumably sterile and ready for immediate use they are resterilized be

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COMMENT

When peripheral nerve lesions follow a regional block procedure the procedure itself must not be automatically and solely condemned until all other possible causes have been considered and eliminated In the greater number of cases the paralysis is not a result of the block procedure or the local anesthetic drug

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fore being injected The vials of Xylocaine solutions the ampules containing the crystals of Pontocaine and Novocain the saline solutions for dissolving the crystals of these drugs as well as *Adrenalin 1 1000 are incorporated in* the block tray prior to its sterilization For autoclaving and cleaning technique as well as other drugs which may be heat sterilized see Chapter 22 page 206

Use Nerve Block with Caution in Patients with Nerve Lesions or Pre Existing Organic Disease—In patients with residues of polio myelitis severed nerves or pre existing or impending pressure on nerves from carcinoma etc nerve block procedures should be used only if the patient accepts the procedure realizing fully that the paralysis existing prior to surgery or that which may occur from the surgery will not be related to the block procedure Whenever these above mentioned conditions exist and block procedures are to be used the disease and the extent to which the peripheral nerves are involved should be noted on the patient s chart by both the attending physician and a consultant

Avoid Irritating Effect of Metal Ions on the Nervous Tissues—Local anesthetic solutions of Xylocaine (lidocaine) and to a lesser extent of Novocain (procaine) have been shown to release copper ions if they come in contact with metal receptacles or metal parts of syringes prior to injection Likewise the tissue damage which may ensue from these ions is evidently caused by the Adrenalin keeping the copper ions in contact with the body tissues for prolonged periods of time Therefore the following precautions should be observed when using solutions of these drugs (1) no Adrenalin should be added to the local anesthetic solution and (2) if Adrenalin is added then with the exception of the needle the equipment used during the block should be glass or stainless steel and 150 T R U (turbidity reducing units) of hyaluronidase should be added to the local anesthetic solution to promote diffusion of the local anesthetic solution (see Figure 2 page 15) Diffusion of the solution will tend to prevent accumulation *of metal ions in any one posi*tion and probably hasten their absorption

TREATMENT

Peripheral nerve lesions in contrast to lesions of the spinal cord and brain, usually repair themselves without treatment in two weeks to six or nine months depending on their severity However in some instances neuritis may persist indefinitely especially if a neurolytic drug has been injected

Immediate Treatment—To relieve pain speed regeneration and assure complete return of function the following may be helpful

Symptomatic Relief of Pain—Aspirin (acetylsalicylic acid), Empirin compound *phenobarbital at night and occasional doses of* codeine may be necessary to relieve the pain of a neuritis

Thiamine Chloride and Nicotinic Acid Therapy—High doses of thiamine chloride (B_1) i e 50 to 100 mg together with high doses of nicotinic acid 25 mg taken orally three to four times a day may facilitate regeneration of the nerve The rationale of this treatment is that thiamine chloride is necessary for nerve function and regeneration while nicotinic acid causes vasodilatation thereby increasing the blood supply to the nerve involved

Sympathetic Block—When the nerves of either the upper or lower extremity are involved a series of stellate ganglion blocks or lumbar sympathetic blocks respectively will increase the blood supply to the involved nerve and has been reported to speed marked recovery of the peripheral nerves[34]

Physiotherapy—Muscle atrophy during the period of regeneration should be prevented by physiotherapy This therapy should include galvanic stimulation of the muscle supplied by the involved nerve as well as massage and exercises

Late Treatment—If the neuritis persists in spite of the above therapy baking of the hypersensitive areas intravenous Novocain or Pontocaine and repeated blocking of somatic or sympathetic nerves with local anesthetic drugs are useful (see Chapter 12 page 120) In the unusual and occasional case of severe neuritis a sympathectomy or a rhizotomy may be necessary Although these two *surgical procedures may seem drastic they are* preferable to opiate addiction

CHAPTER 12

Complications Following the Use of Neurolytic Drugs

WHEN NEUROLYTIC drugs i e alcohol phenol preparations containing propylene and polyethylene glycol oil solutions etc are used the physician performing the regional block procedure automatically takes a known risk regardless of his skill Overflow onto somatic nerves may result in a severe neuritis or in paralysis of an extremity of the diaphragm, or of a vocal cord etc[1] A needle misplaced while performing a paravertebral block a block of the branches of the trigeminal nerve gasserian ganglion block etc with this type of agent may cause a transverse myelitis and/or death[17] Even when small amounts of neurolytic drugs are purposely administered subarachnoidally to treat pain problems e g postherpetic pain and where only a block of the posterior nerve roots is desired unexpected neurological complications such as adhesive pachymeningitis muscular paralysis or paresis of the extremities bladder paralysis rectal incontinence and transverse myelitis and/or a cauda equina syndrome may ensue[2]

Therefore it behooves the physician using neurolytic drugs to be adequately insured and observe the following precautions to avoid legal action in case of an untoward result (1) *explain the procedure and its possible complications to the patient or a member of his family* (2) *have the patient or the responsible member of his family sign a permit for the execution of the block procedure* (3) *do not premedicate or anesthetize the patient for the block procedure* and (4) *if a complication occurs seek the consultation of the necessary specialists i e neurologists internists etc* The third precaution is extremely important for if the patient is not medicated or anesthetized he is clearly consenting to the treatment and is not undergoing any type of therapy which might be objectionable to him The one patient who was blinded at the Mason Clinic (see Figure 18 page 88 and Figure 19 page 89) is a typical example of the efficacy of this precaution—although the patient consulted a lawyer who contacted our insurance company no suit developed and no settlement was made The lawyer for our insurance company was of the opinion the case could be successfully fought on the basis that the plaintiff had willingly submitted to the treatment knowing the possible complications Certainly a block procedure particularly one in which neurolytic drugs are to be used should never be performed against the will of the patient for he may seek retribution on the basis of "technical assault"[18]

The situations most often responsible for difficulties with neurolytic drugs are (1) overflow onto nerves not intended to be blocked (2) corneal ulcers corneal keratitis and ulcerations from block of the gasserian ganglion without overflow (3) transverse myelitis from paravertebral injection and (4) oil embolism

OVERFLOW

Nerves Most Frequently Involved—Nerve block with neurolytic agents is usually limited to block of the sympathetic ganglia the trigeminal (fifth) nerve its ganglia and branches and subarachnoid block of nerve roots as they emerge from the spinal cord Block with neurolytic agents of the peripheral nerves other than the cranial nerves usually results in neuritis and therefore is seldom performed The

32 MOORE D C Complications Following the Use of Efocaine *Surgery* 35 109 114 1954
33 MOORE D C Efocaine—Complications Following Its Use *West J Surg* 61 635 638 1953
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While surgical intervention may appear to be drastic therapy it is to be preferred to opiate addiction

CORNEAL ULCERS, KERATITIS OF THE CORNEA AND ULCERATIONS OF THE SKIN FOLLOWING BLOCK OF THE GASSERIAN GANGLION OR ITS BRANCHES

Perhaps of all blocks of the nerves of the body which are successfully performed with neurolytic agents gasserian ganglion block or block of its branches most often gives satisfactory results Nevertheless in addition to loss of sight and difficult swallowing which have been noted and occur from *misplaced needles* two other complications which may follow a *correctly executed* block of the gasserian ganglion or its branches (the trigeminal nerve) should be discussed

Corneal Ulcers and Keratitis Following Block of the Gasserian (semilunar) Ganglion—Corneal ulcers and/or keratitis are prone to occur following block of the gasserian ganglion because the nerves to the cornea travel through the gasserian ganglion The corneal reflex normally protects the cornea and the eye from trauma caused by drying and/or foreign bodies Its abolishment predisposes the eye to trauma Therefore following block of the ganglion even when a local anesthetic agent is used the eye must be protected for the duration of the block if these complications are to be avoided However when alcohol *is used it is mandatory that the patient* be seen immediately by an ophthalmologist who can instruct him in the correct care of the eye over a period of time

Ulceration, Slough and Gangrene of the Area Innervated by the Terminal Branches of the Maxillary Division of the Fifth Nerve—Following alcohol block or surgical section of *the maxillary division of the fifth cranial nerve* ulcerations and severe slough of areas supplied by its terminal branches have occurred In most instances the area involved has been that innervated by the infraorbital nerve (see Figure 27 page 121) [1] However the one case seen at the Mason Clinic involved the posterior portion of the superior alveolar ridge of the maxillary bone and its surrounding tissues (see Figure 28 page 122)

The cause of this infrequent complication is difficult to determine [21-2?] The maxillary nerve in the cases cited has been either blocked or sectioned at or central to the foramen rotundum Faulty technique direct vascular changes reflex or direct involvement of the sympathetic nervous system and aberrant nerve fibers in or about the gasserian ganglion have all been considered as possible causes but none accepted as fact Macomber [21] and Philpott [2] believe that the immediate cause of the ulceration is probably trauma but they do not try to explain why

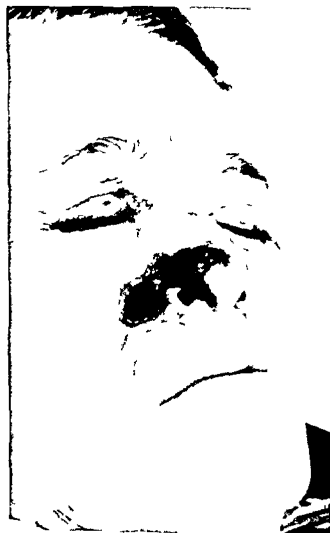


Figure 27 Necrosis of the nose and the cheek following the injection of the maxillary nerve with alcohol for trigeminal neuralgia (contributed by Macomber [21])

nerves most frequently involved from overflow of blocking agents in the procedures listed above are

Branches of the Brachial Plexus—Overflow onto the brachial plexus occurs most frequently following stellate ganglion block phrenic nerve block and infiltration of the scalenus anticus muscle

This results in partial or complete sensory and/or motor loss of the upper extremity

Thoracic and Lumbar Somatic Nerves—Overflow onto these nerves occurs most frequently following block of the thoracic and lumbar sympathetic ganglia or the splanchnic (celiac) plexus

Sequelae of this are partial or complete sensory and/or motor loss of the area supplied by the involved nerve or a severe neuritis

Cervical Sympathetic Nerves or Ganglia—Overflow onto these structures of the sympathetic nervous system occurs most frequently following block of the brachial plexus cervical nerve block thoracic somatic nerve block phrenic nerve block glossopharyngeal nerve block at or slightly distal to the jugular foramen and infiltration of the scalenus anticus muscle A Horner s syndrome i e ptosis enophthalmus and miosis (constricted pupil) ensues

Facial Nerve—Overflow onto this nerve occurs most frequently following infiltration of the skin and subcutaneous tissue prior to performing a mandibular or maxillary block via the mandibular notch It may also follow a glossopharyngeal block distal to the jugular foramen[19]

In such a situation the eyelid cannot be closed and the muscles of the face on the side blocked are paralyzed When a neurolytic drug has been used the prolonged inability to close the eyelid may result in a corneal ulcer and/or a keratitis Here the etiology of the corneal ulcer and/or keratitis is different from that which follows gasserian ganglion block where the loss of the corneal reflex is the responsible factor

Optic Nerve and/or Oculomotor Nerve or Its Branches—Overflow onto these nerves occurs most frequently following gasserian ganglion block and maxillary block if the needle is misplaced If the optic nerve is involved blindness occurs and if the oculomotor nerve or its branches are paralyzed a squint results

Glossopharyngeal Nerve—Overflow onto the glossopharyngeal nerve may occur following an attempt to block the gasserian ganglion if the needle point rests outside the capsule of the ganglion When only the glossopharyngeal nerve is blocked either unilaterally or bilaterally dysphagia is usually the only sign or symptom This complication is the one most often seen following the placement of a neurolytic solution outside the capsule of the gasserian ganglion

Treatment of Overflow—When a neurolytic drug is injected time alone is the best treatment The complication usually corrects itself when the nerve involved completely regenerates The one outstanding exception to this is the optic nerve which may not regenerate once it has been destroyed by alcohol In this instance even if partial regeneration occurs sight often remains impaired

Symptomatic treatment to control pain and prevent atrophy from disuse must be instituted in all cases and the consultation of a neurologist and/or ophthalmologist is invaluable

The treatment of an alcohol or phenol neuritis is not simple Mild cases are treated symptomatically with acetylsalicylic acid Empirin compound phenobarbital or codeine[1] Moderately severe cases may respond to (1) baking the hypersensitive area (2) slowly administered intravenous histamine (2 75 mg dissolved in 500 cc of 5% dextrose in water) twice daily (3) intravenous Novocain (1 gram in 1000 cc of 5% dextrose in water) or intravenous Pontocaine (250 mg in 500 cc of 5% dextrose in water) given slowly twice a day and (4) block of the appropriate portion of the sympathetic nervous system innervating the area of the neuritis for example in case of neuritis of the lumbar nerve lumbar sympathetic block caudal block or spinal epidural block may be used[1] In severe cases of neuritis of this type sympathectomies or rhizotomies may be required[5, 20]

convulsions ensue, and these give way to drowsiness stupor and subsequent coma On the other hand when the heart and lungs are mainly affected, dyspnea cough frothy sputum with occasional hemoptysis cyanosis, precardial pain, dilatation of the heart elevation of temperature and asthmatic or bronchopneumonic type rales result

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Figure 28 Slough of superior alveolar ridge following the injection of the maxillary nerve in the pterygopalatine fossa with 1 cc alcohol to relieve trigeminal neuralgia.

this area is basically susceptible to necrosis and slough However they feel that the sectioning of the nerve renders the skin and mucous membrane in the region insensitive and that the patient is therefore not cognizant that trauma is destroying the tissue Two of Philpotts'[9] cases admitted they "picked" the nose

This complication in relation to the number of times the branches of the fifth nerve are sectioned either surgically or chemically occurs too infrequently to condemn such treatment of trigeminal neuralgia on its account However the seriousness of the complication should make the physician cognizant that he must explain its possible occurrence to the patient prior to the block and warn him that trauma to this area including picking of the nose is dangerous and should be avoided The one patient whose alveolar ridge sloughed following an alcohol maxillary block performed by us should perhaps have been advised to remove her dental bridge after each meal since this bridge hooked onto a single molar in the region where slough occurred

The treatment of these cases is often difficult but includes *antibiotic drugs hot wet packs incision and drainage debridement of the sloughed ulcer and gangrenous area and skin grafting* In instances where the ulcerated area remains infected in spite of the above treatment and/or cannot be prepared for grafting temporary sympathetic denervation by blocking the stellate ganglion may be indicated[1] McKenzie[8] cites one case of slough of the corner of the nose after section of the maxillary nerve for trigeminal neuralgia which could not be grafted until the stellate ganglion had been removed This supports the efficacy of stellate ganglion blocks in these cases to improve circulation to the involved area

TRANSVERSE MYELITIS

Cases of transverse myelitis from the use of neurolytic agents in paravertebral somatic nerve blocks sympathetic ganglion blocks and intercostal nerve blocks have been reported[6, 11, 13] Whenever such agents are injected paravertebrally they may gain access to the spinal cord or subarachnoid space via (1) a misplaced needle (2) a long cuff of dura or (3) the perineural spaces (see Chapter 5 page 48)

OIL EMBOLISM

When using neurolytic solutions containing both oil and local anesthetic drugs great care must be exercised to avoid an intravascular injection from which an oil embolism may result While no cases of this were found in the literature it is assumed that the signs and symptoms would resemble those which occur following the entrance of globules of *fat into the general circulation* Two general groups of signs and symptoms may occur depending on whether the brain or the cardiorespiratory systems are involved[2,7] If the brain is primarily involved loss of memory disorientation irritability and occasionally

of tasting this drug No specific treatment was instituted and no abscess or pneumothorax ensued

Pharynx—Puncture of the pharynx has been mentioned as a possible complication of blocking the carotid sinus nerve [1] We have seen this complication following block of the mandibular nerve and the glossopharyngeal nerve In these instances no problem arose other than that the patient complained of the bitter taste of the local anesthetic solution Should an abscess develop in the neck as it did in Adriani s case cited below it should be treated by the appropriate antibiotic therapy and incision and drainage if necessary

Esophagus—*Adriani et al* [2, 6] *report* two cases of puncture of the esophagus following the direct anterior approach to the stellate ganglion One patient exhibited difficulty in swallowing had a fever of 100 F and exhibited fullness swelling and distinct tenderness on pressure over the area of the puncture Penicillin and streptomycin controlled the infection and the patient had an uneventful recovery To date in over 2 000 blocks performed by the author and his associates using the anterior (paratracheal) approach the signs and symptoms of this complication have not occurred

Kidney, Thyroid, Bladder, Eye, and Peritoneum—It is likely during blocks of the abdomen neck or orbit that adjacent organs are occasionally inadvertently punctured Nevertheless reports of such needle misplacement were not found in the medical literature reviewed

Thoracic Duct—Bonica [3] reports a case of laceration of the thoracic duct while placing a 16 gauge needle prior to inserting a plastic catheter for a continuous stellate ganglion block on the left side He states "Soon after the needle was inserted chyle was seen to extrude around the needle and after removal continued to flow around the catheter for a period of several hours The hazards of using large needles are quite obvious and have deterred us from using this technic in recent years

After this block was discontinued a Horner s syndrome persisted showing that the needle not only damaged the thoracic duct but also the stellate ganglion

Head of the Fetus—Physicians discussing caudal analgesia may mention as a possible complication the inadvertent placement of a caudal needle into the head of the fetus One such case is known to have happened and it resulted in the death of the baby, probably from the injection of 40 cc of the local anesthetic agent but actual reports of it occurring were not found in the medical literature [4]

Penile Fascia—Some urologists warn that impotency may follow the placement of local anesthetic solutions under the deep penile fascia (Buck s fascia) [5] In our small series of 28 consecutive circumcision blocks using a technique of this type we have not had a single case of impotency



Figure 29 Lateral view of needle in place for an alcohol block of the first thoracic sympathetic ganglion The roentgenologist felt considering the position of the patient that the needle was probably in place at the body of the vertebrae Therefore alcohol was injected However the patient coughed immediately and complained of tasting the alcohol The needle was withdrawn and it was concluded that the needle had in reality rested in the trachea No mediastinal abscess or pneumothorax developed

Puncture of Organs and Large Blood Vessels During Block Procedures

THE PUNCTURING of vital organs and large blood vessels during block procedures is always a possibility but with the exception of a toxic reaction to a local anesthetic agent from its intravascular injection sequelae seldom follow The following structures are those most frequently or most likely to be punctured

Blood Vessels —The vertebral femoral and subclavian arteries the abdominal aorta and inferior vena cava are the large vessels most likely to be punctured inadvertently during regional block procedures (see Figure 1 page 14)

Signs and Symptoms —In the normal healthy adult none usually occur if the physician recognizes that he has punctured a vessel and readjusts the needle before injecting the drug But if the drug is injected a generalized systemic toxic reaction may ensue (see Chapter 1 page 16)

On the other hand if the patient has a prolonged clotting time or a blood dyscrasia the ensuing hemorrhage may produce large hematomata and neurologic sequelae even if the needle misplacement is detected and the needle is withdrawn prior to the injection (see Chapter 9 page 89)

Treatment —(See Chapter 1 page 23 and Chapter 9 page 92)

Large or Small Intestine —The rectum or bowel may be punctured while executing a pudendal presacral or a caudal block etc

Signs and Symptoms —When the finger is not in the rectum during a pudendal or presacral block or if a rectal examination is not performed following the insertion of the continuous caudal needle this complication may go unrecognized except that no anesthesia ensues—which could be due to a number of other causes The one other sign that the needle is in the bowel which is most convincing is aspiration of brown semi liquid material—a substance which is peculiar to the contents of the bowel

Treatment —Whether merely withdrawing the needle from the bowel and instituting no further therapy would suffice is difficult to establish Certainly the needle has entered the bowel in some instances without the physician s knowledge and no sequelae have occurred A comparable situation arises if an obstetrician inadvertently places a stitch in the rectum while repairing an episiotomy In such cases although no other therapy except the removal of the stitch is instituted no infection results Nevertheless when the physician performing a regional block knows that the needle has been in the rectum or bowel he is seldom willing to wait and see if infection will result but immediately places the patient on appropriate prophylactic antibiotic therapy It has been our policy in these cases to give both penicillin and streptomycin

Lung —See Chapter 6 PNEUMOTHORAX page 55

Trachea —During the posterior approach to the first thoracic sympathetic ganglion for an alcohol block of this ganglion the author has inadvertently punctured the trachea (see Figure 29 page 125) On injection of the alcohol the patient coughed and complained

CHAPTER 14

Dermatitis

Dermatitis *per se* is a complication which when it occurs by itself, is not catastrophic. On the other hand it may be one of the signs and symptoms heralding or accompanying a systemic allergic reaction to a local anesthetic drug and as such may present itself as part of the picture. While a skin reaction which most often accompanies a severe systemic allergic type reaction is urticarial in nature, that not associated with such a reaction usually manifests itself as a contact dermatitis and usually occurs after numerous exposures to the drug. It is the latter type with which this chapter is predominantly concerned.

Theoretically dermatitis may result in either the physician or the patient from the administration of any of the drugs found in regional block solutions, i.e. local anesthetic drugs, phenol, alcohol, ammonium sulfate, hyaluronidase, propylene glycol, polyethylene glycol, peanut oil, sesame oil, sweet almond oil, solution preservatives (sodium metabisulfite), etc., or combinations of these. However the type of dermatitis to be considered *in detail* in this book is that which follows the use of short acting local anesthetic drugs. Allergies to long acting drugs are infrequent; few reports of such responses could be found in the literature following their use in block analgesia. Then too, the frequency with which solutions with a long duration of action are employed in regional block analgesia, in comparison with local anesthetic drugs, is insignificant.

Dermatitis following the parenteral or topical administration of local anesthetic drugs occurs more frequently than the number of reports in the *present day* medical literature would indicate.[1,31] Lane and Luikart[1] state that dermatitis from local anesthetic agents "is now a matter of common knowledge among dentists and consequently comparatively few cases are reported in the literature at the present." They further point out "Often reports on relatively common diseases appear so seldom in journals that one may conclude from the literature that the condition is relatively unusual."

A review of the literature has revealed the fact that dermatitis frequently affects those who administer drugs used to produce local analgesia. A great number of cases reported in these articles occurred among dentists and physicians, which would indicate that dermatitis may well be considered an occupational hazard of these professions. Gaskill[3] is not convinced that the incidence of dermatitis from Novocain in dentists is as high as the reports indicated. Nevertheless the review by Lane and Luikart[1] of 107 cases of dermatitis from local anesthetics indicates that it is a definite occupational hazard. They found that 83% of cases showing a dermatitis to local anesthetic drugs occurred in either dentists or physicians, dentists constituting the greater number, evidently because of their more frequent exposures and because they seldom if ever wear gloves when performing a block.

ETIOLOGY

The physiological basis for dermatitis from a drug is unknown, but it is a reaction of protection (see Chapter 2, page 33).[36] It is relatively clear from the cases found in the literature as well as the two cases seen by the

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ing The lesion may become eczematous and vesiculated and may ooze It may involve the whole hand or it may be limited to one or two fingers particularly to the interdigital folds (see Figure 30 page 129) If the dermatitis persists and becomes chronic drying scaling and cracking of the skin usually result Exfoliation of the skin occurs leaving it red hypersensitive and painful The rhagades (fissures) which often become deep are extremely painful Probably the most distressing symptoms of contact dermatitis are the pain itching and tenderness

Allergic Reaction of the Dermis —Dermatitis from this cause is infrequent when compared to contact dermatitis and usually follows injection oral or mucous membrane application of the drug In most instances it is a systemic reaction involving the body as a whole and shows itself in toxic erythemas urticaria erythema multiforme generalized morbilliform eruptions and other generalized forms of dermatitis (see Figure 31 page 130 and Figure 32 page 130) Fever lymph node swelling, edema (particularly of the face), swelling of the joints and changes in blood morphology (increase in leukocyte and eosinophil count) often develop Previous exposures to the drug may be difficult to establish definitely

PROPHYLAXIS

Avoidance of the cause of the dermatitis i e the local anesthetic drug is the best therapy for a person who is allergic to local agents and who is interested in remaining comfortable for as Sulzberger and Wise [16] point out "The characteristics of (1) dissemination to distant parts (2) severe pruritis without much dermatitis and (3) a protracted course seem to be encountered frequently in cases of dermatitis due to chemicals possessing local anesthetic properties "

The following precautions may prevent a dermatitis in those physicians who can not avoid the use of a local anesthetic agent to

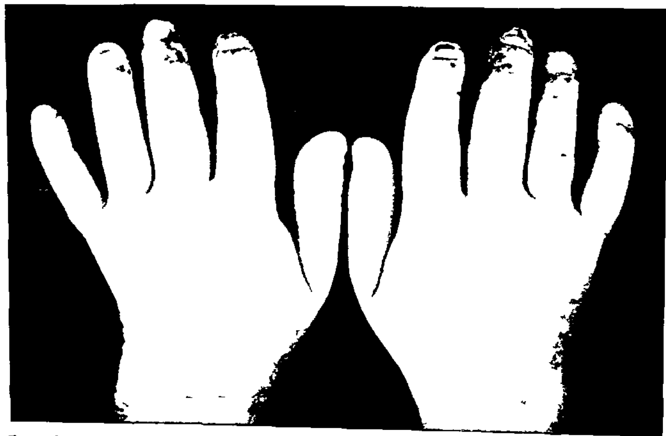


Figure 30 Swelling of the hands following the inadvertent administration of Novocain (procaine) to a doctor with a known allergy to cocaine and its derivatives

author that a person requires many exposures to develop an epidermal allergic reaction to local anesthetic agents.[1,33] However, once sensitized in this fashion, the person will react to the local anesthetic drug in any form or manner of administration. For example, if a person sensitized to Novocain received Novocain in penicillin preparations intramuscularly, in an ointment, intravenously, orally, or otherwise, he would probably suffer a reaction. As a matter of fact, any other local anesthetic drug of the same chemical family as Novocain, i.e., para-amino benzoic acid esters (Benzocaine, Butesin, Butyn, Pontocaine, Larocaine, and Tutocaine), may also cause a reaction in that person if administered parenterally or topically.[28,35,37] Strauss[37] makes the following statements: (1) "Sensitization to an entire group of structurally related compounds may occur as a result of exposure to one of the groups, but this does not necessarily occur." (2) "The question of whether an individual sensitized to one of a group of related compounds is sensitive to all of these compounds must still be determined by patch testing or clinical trial in each individual."

Dermatitis related to the following drugs has been reported in the literature: Apothesin,[1,2] Butesin and Butyn,[3,9,16,17,27] Pontocaine,[10,13,18,23,27,29] Nupercaine,[14,16,20] Benzocaine,[14,27] Novol,[2] Metycaine,[6,34] and Novocain.[15,16,25,29,31,33] A survey conducted by the American Dental Association of their members showed 742 dentists (the number reporting was not given) to be sensitive to one or more items used in their practice.[2] Five hundred sixty-nine reported specific causes for their difficulties relating to local anesthetic drugs as follows: 358 to local anesthetics (no specific one noted), 28 to Butacaine, 20 to Pontocaine, 13 to Benzocaine, and 9 to phenol (these figures do not total 569 but the other causes listed do not directly pertain to anesthetic agents). Since most dentists use Novocain, it may be reasonable to conclude that Novocain is the greatest offender in those cases in which no specific local anesthetic agent was noted.[27] In one dental clinic it was found that 1 in every 12 who handled Novocain developed a dermatitis.[1]

Allergic reactions to phenol are also listed; perhaps this should serve as a warning to investigate thoroughly any dentists on whom a phenol block for a pain problem is being contemplated.

SIGNS AND SYMPTOMS

The signs and symptoms of a dermatitis from a local anesthetic drug depend on whether the epidermis or the dermis reflects the allergy.[39] Templeton[39] made the following important statements: (1) "When the epidermis alone is sensitized, it has generally been brought into this state of sensitization by previous contact of its surface with some external agent capable of producing sensitization." The resulting picture is one of contact dermatitis. (2) "When the dermis alone is sensitized, the offending substance, the allergen, has generally reached it from within the body by way of the blood vessels or lymphatics. The allergen will generally have entered the body by way of inhalation or ingestion." Presumably a subcutaneous or intramuscular injection could be responsible in sensitizing the dermis.

Allergic Reaction of the Epidermis (Contact Dermatitis).—Contact dermatitis, i.e., an allergy of the epidermis, is the type of dermatitis due to local anesthetic drugs more frequently seen.

Contact dermatitis usually has an incubation period, and the symptoms often do not appear for days, weeks, or even years. The affected area is usually limited to that part of the body which comes in contact with the drug. Therefore, since many of the dermatitides to local anesthetic drugs are occupation acquired, the areas usually affected are found on the hands, forearms, face, and neck, i.e., the exposed areas.

The characteristics of a contact dermatitis may vary, and one or all of the following may occur in the same patient. Usually the acute signs and symptoms are those of irritation, i.e., reddening of the skin, swelling, and itch

block is completed even if he is not at that time allergic to the drug. Without this precautionary washing off of the drugs, repeated exposure may eventually result in an allergy.

Use a Local Anesthetic Agent of a Different Chemical Structure.—If a physician or dentist becomes allergic to one chemical group of local anesthetic drugs, other agents of a different chemical structure may be safely used (see Table VII, page 20). For example, he may be allergic to the para-amino benzoic acid esters (Novocain, etc.) but might be able to come in contact with the benzoic acid esters (Metycaine, etc.) without provoking a contact dermatitis.[28, 37] Whether this is true or not should be determined by patch tests rather than clinical trial.

Patch Test.—As pointed out in previous chapters, this test may be of little value in determining whether or not a patient will have a high blood level type or a "clinical anaphylactic shock" type of systemic reaction to a local anesthetic drug administered parenterally. However, most of the authors cited in the bibliography to this chapter agree that the patch test is reliable in determining whether a dermatitis is or is not caused by a local anesthetic drug. Laden and Wallace[29] when testing ten dentists with an allergy found by patch tests that nine of them showed an eczematous reaction to local anesthetics.

While patch tests usually become positive within 24 to 48 hours, if at all, negative results should be examined for several subsequent days up to a week or longer.[30]

TREATMENT

The management of a dermatitis depends on the type of lesion. However, the one treatment which is the most effective and is applicable irrespective of the type of lesion is *removal of the offending drug*.[40, 47] It has been the author's and his associates' custom not to treat dermatological lesions but to refer them to the Clinic's dermatologist. The following treatment, part or all of which is used at the Clinic, is not original but obtained from the experience of many workers in this field.[40, 48]

Eczematous and Eczematoid Dermatitis.—The rules of therapy when treating an eczematous or an eczematoid dermatitis are: (1) a few well chosen remedies are preferable to the haphazard use of many; (2) shotgun prescriptions are to be avoided as they may contain drugs which may aggravate the allergy; (3) treatment must be conservative and not over-aggressive, i.e., "Primum non nocere" (above all do no harm); and (4) exact means of a drug's application must be described—the patient must not merely be told to "go home and use it."

Relief of itching.—Itching is probably the most common complaint of a contact dermatitis. Treatment consists of:

I. *Antipruritics.*—The effects of calcium gluconate, sodium thiosulfate or strontium bromide given intravenously have proven disappointing. The antihistamine drugs parenterally, i.e., Benadryl (diphenhydramine hydrochloride) 50 mg. orally or intravenously or Pyribenzamine (tripelennamine hydrochloride) 100 mg. orally every 3 to 4 hours, will in most instances control the pruritis. If these drugs are unsuccessful, then the other treatments listed below may be used. While intravenous and oral uses of these drugs are very effective, the external use of the antihistaminic compounds to date has not proven satisfactory.

II. *Sedation.*—Barbiturates, salicylates and chloralhydrate are preferable to the opiates which tend to aggravate itching, although in some cases they give excellent relief. Phenobarbital in ¼ to ½ gr. (15 to 30 mg.) doses three times a day and at bedtime is usually satisfactory.

III. *Splint and wrap part.*—This avoids subconscious scratching and may be extremely helpful.

IV. *Avoid soap.*—Since soap may exaggerate dermatitis, starch, bran, oatmeal or potassium permanganate baths are to be preferred to cleanse the skin and soothe the irritation.

V. *Dehydration.*—In some instances this will markedly relieve itching and may be accomplished by limiting fluid intake and by the use of diuretics.

Figure 31 Erythematous reaction in a patient allergic to Novocain (procaine) 7 hours after raising 2 skin wheals on left forearm with 1.0% Novocain (contributed by Chipps*)



Figure 32 Circumorbital and right cheek edema in the same patient shown in Figure 31 following failure to take antihistamine 24 hours after raising the skin wheals on the left arm and 7 days after extraction of upper right first molar under Novocain anesthesia (contributed by Chipps*)

which they react in this fashion and may prevent this type of allergy in a physician who has not yet developed it

Care of Physician's Hands During Administration—Gloves should be worn when performing blocks to prevent contact with the local anesthetic agent being employed This *is singularly important if a large number of* regional procedures are performed or if the physician has an allergy to the local anesthetic agent of his preference If a physician has already developed an allergy from handling local anesthetic drugs and cannot avoid their use protective creams such as Covicone (combination of Silicone Nitrocellulose and castor oil in a greaseless vanishing cream base) applied to the areas of the skin of the forearm not covered by the gloves may give added protection

Should the local anesthetic drug come in *contact with the physician's skin in areas that* have not been protected by gloves or creams the area should be washed as soon as the

ministration was used and lowest when the drug was given orally. This would indicate that repeated spillage of the drug onto the skin of the personnel administering it was responsible for the dermatitis.

Chlorpromazine has been used in regional anesthesia by the author and his associates for sedation following regional nerve blocks other than those which in themselves lower the blood pressure (spinal, epidural and celiac plexus block). Since the drug has been given intravenously in these cases, it is possible that the solution could on occasion come in contact with the skin. This might result in allergy and indicates the necessity of washing the hands when spillage occurs.

REMEMBER, GLOVES ARE NOT USED WHEN INJECTING LOCAL ANESTHETIC SOLUTIONS ONLY TO ASSURE ASEPSIS, WHICH MAY BE OBTAINED BY CAREFUL SCRUBBING OF THE HANDS, BUT FOR THE PROTECTION OF THE PHYSICIAN OR DENTIST AGAINST THE OCCUPATIONAL HAZARD OF AN ACQUIRED ALLERGY TO LOCAL ANESTHETIC AGENTS USED IN THEIR DAILY PRACTICE.

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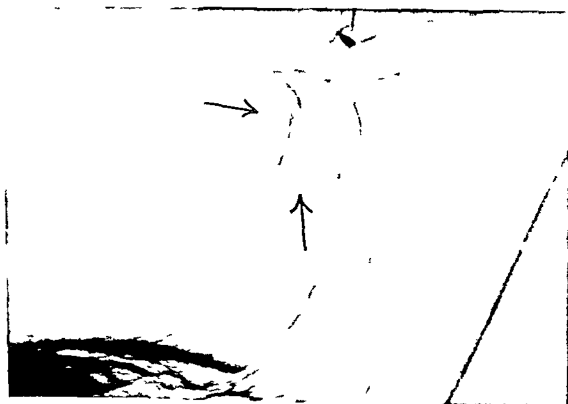


Figure 33. Dermatitis from tincture of merthiolate used to prepare the skin prior to a stellate ganglion block. During the past 12 years the author has used this solution to prepare the skin and this is the only time this type of complication has occurred.

Local therapy —Local therapy is indicated when the lesion progresses past the stage of simple itching It consists of the following generalized treatment

I In acute erythematous and vesicular stages wet dressings (solution of aluminum acetate etc) are usually the treatment of choice If wet dressings are not practical then lotions pastes and emulsions may be used and powders are helpful in intertrigi nous areas such as the groin under the breasts and between the toes

II In subacute stages with dry crusted scaly areas lotions salves and x ray treat ment are preferred

III In chronic dry thickened lichenfied scratched eczematoid dermatitis treatment is aimed at (1) relief of scratching by roent gen ray and antipruritics and (2) relief of the thickening of the skin by tars and other preparations

Cortisone Therapy —Cortisone orally has its merits in treating contact dermatitis It may stop the itching and also improve the lesion Hydrocortisone ointment may be of help in treating localized contact dermatitis It may also be given orally After an initial dose of 100 mg is taken by mouth 50 mg is taken every 4 to 6 hours for 6 to 8 doses and finally every 6 hours for 4 doses [47] Cortisone taken over protracted periods can be dangerous (see Page 254)

A C T H (corticotropin) may have the same beneficial effects on dermatitis as corti sone

Urticaria and Urticarial Dermatoses —This type of reaction seldom persists long enough when caused by a local anesthetic agent to warrant treatment such as autohemotherapy ichthyol alkalis or dilute hydrochloric acid by mouth large doses of reduced ferric iron with ammonium citrate and oral peptones —therapy which proves useful in food aller gies However when urticaria occurs follow ing the use of a local anesthetic agent it should be treated immediately to prevent pro gression to generalized edema particularly of the larynx where it may result in respiratory obstruction Treatment by the anesthesiolo gist to prevent this consists of the immediate use of one or more of the following Ample time i e 10 to 15 minutes should be allowed for one type of therapy to become effective before trying another

Intramuscular Adrenalin (epinephrine) ¼ cc of 1 1 000 solution

Intravenous antihistaminic drugs i e 50 mg of Benadryl etc given slowly These drugs usually produce satisfactory results and may be repeated every 3 hours if angioneu rotic edema recurs We have emphasized that larger intravenous doses of Benadryl are sel dom more effective and in addition may re sult in untoward reactions (see Chapter 2 page 34) In the few cases of urticaria seen by the author this therapy was most effec tive

Intravenous calcium gluconate (10 cc of a 10% solution) or *calcium chloride* (5 to 10 cc of a 5% solution) given *slowly* may achieve rapid cessation of the urticaria but the re sults are not as consistent as with the anti histaminic drugs If calcium chloride is used it is mandatory that it be given very slowly i e not over 2 cc per minute or cardiac syn cope may occur

COMMENTS

Dermatitis from Solutions Used To Prepare the Skin —Dermatologists report it relatively frequently and state that mercurials rate as one of the most potent sensitizing agents (see Figure 33 page 133) [14 16] We include men tion of it here to emphasize the point that the local anesthetic drug is not always responsible for a dermatitis Whenever a dermatitis oc curs a patch test to determine the offending drug should be made and the patient should be told the results so that he can warn another physician who might undertake another block procedure in the future

Dermatitis from Chlorpromazine —Contact dermatitis following the use of chlorpromazine in personnel administering the drug has been reported [49] The incidence of dermatitis was highest when the intramuscular route of ad

ministration was used and lowest when the drug was given orally This would indicate that repeated spillage of the drug onto the skin of the personnel administering it was responsible for the dermatitis

Chlorpromazine has been used in regional anesthesia by the author and his associates for sedation following regional nerve blocks other than those which in themselves lower the blood pressure (spinal epidural and celiac plexus block) Since the drug has been given intravenously in these cases it is possible that the solution could on occasion come in contact with the skin This might result in allergy and indicates the necessity of washing the hands when spillage occurs

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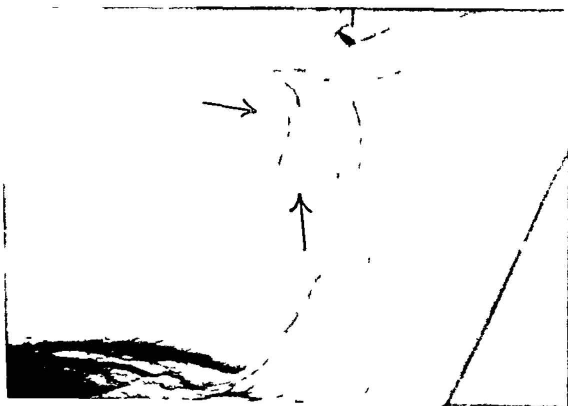


Figure 33 Dermatitis from tincture of merthiolate used to prepare the skin prior to a stellate ganglion block During the past 12 years the author has used this solution to prepare the skin and this is the only time this type of complication has occurred

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PART II

COMPLICATIONS OF SPINAL (SUBARACHNOID) BLOCK AND EPIDURAL (PERIDURAL) BLOCK

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Introduction to Part II

The complications discussed in this part of the book are those which are primarily encountered following spinal (subarachnoid) or epidural (peridural) blocks. The term "spinal (subarachnoid) block" is clear to all physicians. However, the term "epidural (peridural) block" perhaps should be clarified as it is used herein. Epidural analgesia is produced by placing the local anesthetic solution in the triangular or rounded space which is situated between the dura mater on one side and the lining of the vertebral canal (the ligamenta flava, the lamina and pedicles of the vertebrae, and the posterior longitudinal ligament) on the other side. Spinal epidural block refers to the placement of the local anesthetic solution into the epidural space via the thoracic or lumbar region. On the other hand, caudal block refers to the placement of the solution into the sacral portion of this space via the caudal canal.

It must be remembered that any of the complications found in Part I may develop, since local infiltration is often performed as a preliminary step to tapping these two spaces, and that those complications which are incidental to regional block procedures and found in Part III may likewise occur.

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It must be remembered that any of the complications found in Part I may develop since local infiltration is often performed as a preliminary step to tapping these two spaces, and that those complications which are incidental to regional block procedures and found in Part III may likewise occur.

Hypotension from Spinal or Epidural Block During the Induction of Anesthesia or During the Operative Period

VASCULAR HYPOTENSION may occur following any method of producing regional analgesia It is frequently seen following purposely administered spinal and epidural procedures but seldom accompanies peripheral nerve blocks or local infiltration unless a systemic toxic reaction to the local anesthetic agent ensues or a misplaced needle results in a spinal block an epidural block severe hemorrhage or a severe pneumothorax

Hypotension *per se* when *purposely* induced to effect hypotensive anesthesia may not be detrimental[11] However if unexpected hypotension goes unrecognized or is inadequately treated it may lead to severe hypoxia and rapid deterioration of the patient s condition with one or more of the following thrombosis cerebral hypoxia renal failure convulsions cardiac failure and death Therefore when an unexpected hypotension occurs during regional block procedures it demands immediate attention

Hypotension and oxygen want during spinal and epidural block go hand in hand and to treat one the physician must be cognizant of the cause and treatment of the other Therefore this chapter and the next two should be considered together

ETIOLOGY

The arterial pressure depends on (1) the pumping action of the heart (2) peripheral resistance (3) quantity of blood in the arterial system (4) viscosity of the blood and (5) elasticity of the arterial walls[13] Therefore any alterations of these by a regional anesthesia may cause hypotension unless the body rapidly adjusts to the change Of the regional block techniques used daily spinal and epidural blocks are the ones most likely to alter the cardiovascular system

The hypotension which occurs during spinal and epidural analgesia results *primarily* from paralysis of the nerves to the regions anesthetized Not only are the sympathetic and somatic nerve fibers paralyzed resulting in peripheral vascular dilatation with pooling of the blood and decreased respiration respectively but the nerves to those organs which normally maintain the blood pressure as well as those which normally compensate for hypotension are also paralyzed In addition to these causes of hypotension after spinal and epidural block other causes of hypotension not directly dependent on the paralysis of the nerves to the dermatomes are (1) systemic toxic reaction (2) surgical manipulation (3) positional changes (4) certain diseases (5) hemorrhage (6) pregnancy and (7) premedication particularly chlorpromazine

Alterations in the Cardiovascular System from Paralysis of the Nerves to the Dermatomes, i e, Neurogenic Circulatory Depression—This is the *principal* cause of a hypotension which follows a spinal epidural or splanchnic (celiac plexus) block The circulatory changes which occur are due to changes in the venous as well as the arterial side of the vascular system[14,16] Paralysis of the sympathetic vasoconstrictor fibers to the arterioles results in dilatation of the arterioles and a

decrease in the peripheral resistance Likewise there is an actual dilatation of the peripheral veins with a pooling of blood in the venules Paralysis of the sympathetic fibers of the splanchnic (celiac) plexus alone is not singly responsible for the fall in blood pressure seen with spinal and epidural block but may be a contributing factor It has been shown in dogs that bilateral splanchnicectomy alone does not produce the characteristic blood pressure fall of spinal anesthesia[14]

As a result of the paralysis of the sympathetic fibers a normal circulatory system is suddenly changed by vasodilatation into one which is too large for the circulating volume of blood To compensate the vascular bed in the unanesthetized area constricts usually resulting in a shift of the blood away from the heart and lungs to the splanchnic area and the lower extremities The height of the analgesia following spinal or epidural block directly governs the degree of this shift and in general when the level rises above the umbilicus (T_{10}) there is a progressive loss of the capacity for reflex vascular compensation[14, 1, 18] In other words as the paralyzed area increases in size the area in which the blood vessels can constrict to decrease the size of the total circulatory system is reduced Consequently the blood volume becomes insufficient in relation to the size of the vascular system to maintain an adequate blood pressure

The loss of function of portions of the sympathetic nervous system is probably the primary reason for alterations in the cardiovascular system but lowered blood volume paralysis of the skeletal muscles and paralysis of the nerves to the kidneys and adrenal glands have been mentioned as factors which may contribute significantly to the hypotension when the nervous system is chemically sectioned by a local anesthetic agent

Lowered Blood Volume—When a fall in blood pressure from spinal block or epidural block occurs in a patient whose blood volume is low the hypotension may be quite difficult to correct Nicholson[19] states "The most alarming blood pressure falls associated with operations in the upper abdomen under spinal anesthesia have been experienced in those patients suffering from diseases of the gastrointestinal tract associated with a large loss of body weight over a short period of time Investigations of our own as well as of others have shown this group of patients to be suffering from marked reductions of their total blood volume[20, 1] Unfortunately these reductions in blood volume are not brought to light by the use of the routine concentration tests such as the hemoglobin red blood cell count and hematocrit When the Evans blue dye method of blood volume determination has been employed and the deficit corrected these patients will tolerate spinal anesthesia for operations in the upper portion of the abdomen"

Paralysis of the Skeletal Muscles—Normally the skeletal muscles act as a support for the vascular system and perform a muscular milking action aiding blood return to the heart In addition the muscles of respiration play an important part in the return of the blood to the heart because the decrease in the intrapleural pressure during inspiration acts as a suction pump to bring blood into the great veins Therefore when the skeletal musculature is paralyzed by spinal or epidural block the venous return is decreased resulting in decreased venous pressure increased venous circulation time increase in right auricular pressure reduced cardiac output and a decline in blood pressure[14 16]

Seevers and Waters[23] state" In view of experimental and clinical observations we believe that too little stress has been placed on the role of respiration in the maintenance of cardiovascular integrity during spinal anesthesia Our conception of the train of events occurring during untreated high spinal block is as follows initial decrease in peripheral resistance to blood flow by vasomotor nerve and skeletal muscle paralysis decrease in minute volume respiration accompanying intercostal nerve paralysis inadequate oxygenation of blood diminished minute volume blood flow progressive loss of vascular tone over the whole body and acute cardiac incompetence, both the result of oxygen deprivation

failure of the medullary respiratory mechanism as the nutrient flow of blood becomes inadequate the latter occurring while the heart is still capable of being revived by oxygenation"

Paralysis of Nerves to Adrenal Glands and Kidneys—Paralysis of the nerves to the adrenal glands and kidneys has been mentioned as a cause for hypotension following spinal or epidural block [14, 18, 24] While perhaps contributory, it is doubtful that this mechanism is the inciting factor

Systemic Toxic Reactions to the Local Anesthetic Drug—Hypotension is seldom caused in spinal analgesia by a systemic reaction to the local anesthetic drug itself because the quantity of the drug used is relatively small However, in epidural analgesia where larger quantities of local anesthetic solutions are injected, it is possible although not likely that an ensuing hypotension is the result of a systemic toxic reaction (see Chapter 1 page 6 and Chapter 2 page 33)

In over 10 000 epidural blocks and spinal blocks performed at the Mason Clinic there have been 9 convulsions following epidural blocks (1 in lumbar area 8 in caudal area) and none following spinal blocks These convulsions were treated immediately with oxygen by bag and mask and no drop in blood pressure was registered However if treatment is not instituted in such cases cardiovascular collapse with a fall in blood pressure may follow

Surgical Manipulation or Other Forms of Trauma, i e, Injuries, Hemorrhage etc—In over 10 000 spinal and epidural blocks, we have observed that when the height of anesthesia rises to T_4 and blocks the major portion of the nerve supply to the dermatomes the blood pressure often does not fall until intra abdominal manipulation starts Collins [4] states "In human experiments 200 mg of procaine were injected and high spinal anesthesia obtained to the level of C_7 (7th cervical segment) No hypotension or complications of significance developed Hypotension did develop when surgical manipulation began and when the manipulation was maximal the hypotension was maximal The patients were unable to bring their pressures back to normal levels It is worth noting that patients who have no disease other than the immediate problem for which they are being treated and who have hypotension immediately following spinal or epidural block will often compensate with no therapy other than oxygen provided surgical manipulation does not occur prior to re establishment of a normal pressure However if surgery is started prematurely, it may be difficult or in rare instances impossible to raise the blood pressure even by means of vasoconstrictor drugs or fluid replacement therapy

Positional Changes—It is common knowledge among anesthesiologists that in the anesthetized patient a change in position particularly a rapid one may result in hypotension and in the occasional instance death [14, 25] Such a result may take place under any type of anesthesia but is more prone to occur under spinal or epidural block

Soffer and Sweet [25] summarize their experience as follows "Significant gravitational movement of blood in the legs occurs following minor position shift during the state of vasomotor paralysis produced by certain autonomic blocking drugs high or low spinal anesthesia, and to a lesser extent during thiopental sodium anesthesia Depression or elevation of the lower extremities may therefore be utilized to lower or raise the subject's blood pressure as the situation demands One must however be constantly aware of the drastic circulatory changes that may rapidly ensue if these position shifts are produced or resolved suddenly and without continuous observation Three cases are described that illustrate the blood pressure changes that may occur during spinal anesthesia as a result of the assumption or resolution of the lithotomy position The fatal outcome of case 3 was directly related to such a postural shift of the lower extremities"

Miscellaneous Causes of Hypotension—During spinal and epidural block the following may cause hypotension While they may

occur during the block procedure they are not necessarily directly associated with the drug used, paralysis of the nerves to the dermatomes, surgical manipulations, or postural changes

Coronary Occlusion, Pulmonary Embolus, Cerebral Vascular Accident, Pulmonary Collapse or Cardiac Failure.—These events may occur during regional block procedures and one usual sign of them is a severe hypotension. In some instances these accidents may be directly attributed to hypoxia, hypotension, surgical manipulation, position, previous disease, etc., but they may sometimes occur without discernible cause

Hemorrhage.—If hemorrhage occurs prior to or during surgery, then hypotension results from surgical shock—of course this may happen under regional anesthesia just as well as under general anesthesia. Often when hemorrhage occurs spontaneously, as in ruptured ectopic pregnancy, incomplete abortion, etc., the patient will have an immediate hypotension but will usually compensate for it and over the next few hours the blood pressure may return to normal with the help of replacement therapy. When a normal or near normal blood pressure is obtained, the patient may tolerate a spinal or epidural block well. If, however, a surgical procedure is decided upon before this return to normal and a spinal or epidural anesthesia performed, the lowered blood volume plus the block procedure may result in a hypotension that is difficult or impossible to correct

A blood pressure fall from hemorrhage is a more serious problem than that from a spinal or epidural block. Burch and Harrison[16] state: "From the data it appears that in spinal anesthesia the initial change is, as would be expected, in arterial pressure and that venous return and output of the heart are affected secondarily. This sequence of events is just the opposite of that in hemorrhage. In this condition Blalock has shown that the cardiac output decreases first and is already reduced by from one third to one half before a significant decline in mean arterial pressure occurs. These observations probably explain the well recognized clinical fact that a low blood pressure due to hemorrhage is a much more serious sign than a similar blood pressure fall in a patient under spinal anesthesia. . . ." However, if a blood pressure fall occurs from both hemorrhage and the effects of the anesthetic, the problem is singularly precarious and this is probably the outstanding reason why spinal and epidural blocks are usually contraindicated in a patient who has suffered a severe hemorrhage prior to surgery and whose blood pressure is still low

Pregnancy.—Not uncommonly, when giving a pregnant patient at term an anesthetic, the blood pressure falls regardless of the method used. According to Eckenhoff[76] and Wilkening *et al*[77] this is due to the fact that when the patient is placed supine, a position most pregnant patients avoid, the gravid uterus falls back occluding the vena cava—this decreases the return of blood to the heart, which in turn reduces the cardiac output and a fall in blood pressure results

It must be cautioned that whenever the systolic blood pressure of a pregnant woman drops below 80 mm, steps to raise the blood pressure should be instituted, otherwise the baby may suffer chronic hypoxia or anoxia, probably because a systolic pressure of over 80 mm is usually necessary to assure circulation of blood through the uterus during its contraction

Chlorpromazine (Thorazine, Largactil) as Premedication.—At the present time the clinical use of chlorpromazine is still in its experimental stages and further evaluation of the drug is necessary before conclusive statements about it can be made. It has been *our observation* at the Mason Clinic that when the drug is used for its sedative effect *prior to* or immediately following a peripheral nerve block, some degree of hypotension may ensue. Such a hypotension is seldom a problem. But if this drug is given *before or during* a spinal block, an epidural block, or a splanchnic block, a severe hypotension may develop. In this case the low blood pressure is of such a nature that it seldom *adequately* responds to the usual dosage of the common vasoconstric

tor drugs e g ephedrine sulfate Neosynephrine Vasoxyl or Levophed While Levophed or Neosynephrine if run rapidly may somewhat elevate the pressure stopping them before either the regional block or the dosage of chlorpromazine has been dissipated may again lower the blood pressure to a precarious level Foster *et al* [27] after studying the effects of Levophed and epinephrine on the blood flow in the hands of patients who had previously received intravenous doses of chlorpromazine concluded that chlorpromazine decreases the pressor effect of Levophed and reverses the vasoconstrictor effect of epinephrine These controlled experiments seem to substantiate our clinical observations Foster *et al* [27] also concluded that the fall in blood pressure from chlorpromazine is due to its powerful vasodilatory action which is a result of both the central and the local effects of the drug

Therefore at the Mason Clinic chlorpromazine has not been used with regional block procedures for sedation in (1) patients who are to receive nerve blocks which in themselves lower blood pressure such as celiac plexus block spinal block and epidural block (2) patients who have hypertension (3) the elderly and (4) patients who have had coronary occlusions Furthermore because most of our block patients receive an opiate i e morphine sulfate 1/6 to 1/4 gr (10 to 15 mg) combined with a belladonna drug i e atropine or scopolamine 1/150 to 1/100 gr (0.4 to 0.6 mg) 30 to 40 minutes before coming to surgery we have not exceeded an initial dose of 20 mg of chlorpromazine intravenously in any patient and our usual dose is 10 to 15 mg Only in the *occasional* patient has it been necessary to add 5 or 10 mg to this usual dosage to obtain the desired results At least 15 to 30 minutes should elapse before the initial dose of chlorpromazine is supplemented By observing these precautions we have been able to obtain satisfactory sedation with chlorpromazine yet avoid one of its serious effects—excessive hypotension

When chlorpromazine is employed for preoperative sedation by the Anesthesia Department at the Mason Clinic, it is given intravenously by one of the physicians in that Department In those patients receiving regional block procedures *which do not require paresthesias* for a successful block e g., local infiltration and certain peripheral nerve blocks (cervical block etc) the chlorpromazine is usually administered in the above noted fashion *one half an hour prior to the block* In those patients receiving blocks *which require that paresthesias be elicited* for a successful block e g brachial plexus block sciatic nerve block etc it is administered immediately upon completion of the block It is interesting to note that the drug has a retrograde amnesic effect Often when it has been given following the elicitation of paresthesias the patient, when seen on postoperative rounds does not remember the "electric shock like" sensation caused by stimulating a nerve

It must be remembered that (1) there is no effective antidote to chlorpromazine at the present time (2) the drug is excreted slowly and (3) the depressing effects on the cardiovascular and respiratory systems may remain for considerable periods after the operation Therefore the drug must be administered with care Furthermore every patient who is to be operated on under spinal block epidural block or intercostal deep splanchnic (celiac plexus) block or in whom one of these blocks is to be used in therapy or diagnosis must be asked if he has been on chlorpromazine therapy The order sheet and nurse's notes must be checked immediately before performing the block These warnings are emphasized by one death and two near fatalities which have occurred recently because these precautions were not exercised

In one case of Bonica's [28] the surgeon had seen the patient at home and placed her on chlorpromazine because of nausea associated with an intestinal obstruction The surgeon did not mention this nor did the nursing notes have any record of it The patient was given an epidural block for surgery and a severe hypotension developed which was difficult to correct and although the patient survived the surgical procedure she succumbed in the im

mediate postoperative period probably from the prolonged period of hypoxia during surgery

The second case was one of Owens's.[2] He saw the patient on preoperative rounds at four o'clock in the afternoon. Later the surgeon visited the patient and found him quite disturbed concerning the operation, and therefore he ordered 25 mg. of chlorpromazine immediately and another 25 mg. at midnight. At seven o'clock the next morning, without a review of the chart, a spinal anesthetic was administered with light Nupercaine to T_4. The patient would not respond to questioning, and the blood pressure had fallen from a preoperative level of 90/60 to one which was unobtainable. Four mg. of Vasoxyl was given intravenously and no response was noted. This was followed by 3 mg. of Neosynephrine and the blood pressure slowly crept back to 70/40. The patient's blood pressure was sustained for the rest of the operation and in the immediate postoperative period by an unusually concentrated Neosynephrine drip (40 mg. of Neosynephrine in 1,000 cc. of 5% dextrose in distilled water). The patient's convalescence was uneventful, and there was no untoward result from the period of hypotension and the associated hypoxia.

In the third case, a patient of the author, had carcinoma of the common bile duct and was having severe intractable pain. The internist called and made an appointment for the patient to have a splanchnic (celiac plexus) block. She had been placed on chlorpromazine 25 mg. t.i.d. to help control pain. However, this was not called to my attention and was not recorded on the chart when I first visited her. Three hours later when she was brought to the block room the chart was not reviewed. In the meantime the fact that the patient was receiving chlorpromazine had been recorded on the order sheet and the nurse's notes. As is our custom in these cases a block is performed with a local anesthetic agent so that the degree of pain relief obtained may be evaluated prior to the use of the alcohol. Immediately after the block the patient's blood pressure dropped from a preoperative level of 130/80 to 45/0. In this instance 5 mg. of Vasoxyl intravenously raised the blood pressure to 70/40, but it fell again to 45/0 within 10 minutes. It was then again raised and maintained at 90/60 with a Neosynephrine drip (10 mg. in 1,000 cc. of 5% dextrose in distilled water). The heart rate was grossly irregular. The patient was rapidly digitalized and made an uneventful recovery. Immediately after the treatment of the hypotension I remarked to my resident that the patient looked as if she had been receiving chlorpromazine. The chart was checked and I was quite chagrined to find that this had been the case.

SIGNS AND SYMPTOMS

The signs and symptoms of hypotension in spinal and epidural block are related to changes in the cardiovascular system resulting from decreased circulation of blood (stagnant hypoxia) and to respiratory embarrassment (tidal hypoxia).

Fall in Blood Pressure.—Properly speaking hypotension is a fall in arterial pressure below a patient's recognized normal. Therefore a specific arterial level of 70 or 80 cannot be chosen as a standard for all patients. An "on the spot" analysis by the physician who takes into consideration the physical status of the patient and other factors, must determine whether hypotension exists. For example, a healthy but obviously nervous patient with a preoperative pressure of 180 mm. systolic and 100 diastolic may, after a quiet night of bed rest, have a pressure of 130 mm. systolic and 90 mm. diastolic. During a spinal or epidural block procedure his pressure may drop to 90 mm. systolic and 60 mm. diastolic, but if he responds rationally to questioning and states that all is well, if his skin is warm and dry, his respirations are adequate and he has no change in his heart rate and rhythm, then his hypotension should not cause alarm. Correction of it other than continual close observation for further drop is unnecessary.

On the other hand, a diabetic with associated arteriosclerotic heart disease whose

tor drugs, e.g., ephedrine sulfate, Neosynephrine, Vasoxyl or Levophed. While Levophed or Neosynephrine, if run rapidly, may somewhat elevate the pressure, stopping them before either the regional block or the dosage of chlorpromazine has been dissipated may again lower the blood pressure to a precarious level. Foster *et al*[illegible] after studying the effects *of Levophed and epinephrine on the blood flow* in the hands of patients who had previously received intravenous doses of chlorpromazine concluded that chlorpromazine decreases the pressor effect of Levophed and reverses the vasoconstrictor effect of epinephrine. These controlled experiments seem to substantiate our clinical observations. Foster *et al*[27] also concluded that the fall in blood pressure from chlorpromazine is due to its powerful vasodilatory action which is a result of both the central and the local effects of the drug.

Therefore, at the Mason Clinic, chlorpromazine has not been used with regional block procedures for sedation in (1) patients who are to receive nerve blocks which in themselves lower blood pressure, such as celiac plexus block, spinal block and epidural block, (2) patients who have hypertension, (3) the elderly and (4) patients who have had coronary occlusions. Furthermore, because most of our block patients receive an opiate, i.e., morphine sulfate 1/6 to 1/4 gr (10 to 15 mg), combined with a belladonna drug, i.e., atropine or scopolamine 1/150 to 1/100 gr (0.4 to 0.6 mg), 30 to 40 minutes before coming to surgery, we have not exceeded an initial dose of 20 mg of chlorpromazine intravenously in any patient and our usual dose is 10 to 15 mg. Only in the *occasional* patient has it been necessary to add 5 or 10 mg to this usual dosage to obtain the desired results. At least 15 to 30 minutes should elapse before the initial dose of chlorpromazine is supplemented. By observing these precautions we have been able to obtain satisfactory sedation with chlorpromazine, yet avoid one of its serious effects—excessive hypotension.

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It must be remembered that (1) there is no effective antidote to chlorpromazine at the present time, (2) the drug is excreted slowly and (3) the depressing effects on the cardiovascular and respiratory systems may remain for considerable periods after the operation. Therefore, the drug must be administered with care. Furthermore, every patient who is to be operated on under spinal block, epidural block, or intercostal deep splanchnic (celiac plexus) block or in whom one of these blocks is to be used in therapy or diagnosis must be asked if he has been on chlorpromazine therapy. The order sheet and nurse's notes must be checked immediately before performing the block. These warnings are emphasized by one death and two near fatalities which have occurred recently because these precautions were not exercised.

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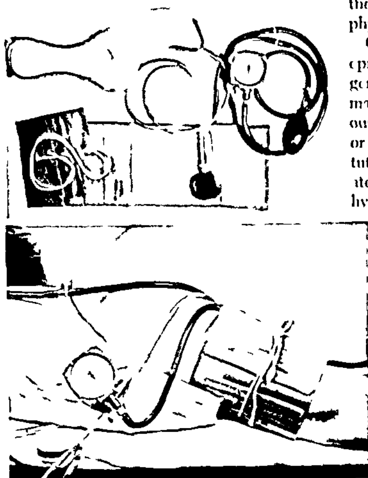


Figure 34 (A) Blood pressure cuff with the chest piece of the stethoscope sewn in place so that it will not move Hernia cord has been stitched to the cuff so that when the cuff has been placed on the arm the hernia cord may secure it in place (B) Cuff applied to arm Note that the hernia cord functions to prevent slipping of the cuff during motion

constrictor drug prior to a block has been voiced on two bases that in a large number of patients particularly if the spinal or epidural block remains below the umbilicus (T_{10}) no fall in blood pressure occurs and that if the patient is hypertensive the use of a vasoconstrictor prior to a blood pressure fall may elevate the pressure and cause blood vessel rupture However in over 10 000 spinal and epidural block procedures performed at the Mason Clinic in the past eight years no difficulty has been encountered from use of vasoconstrictor drugs prior to spinal and epidural blocks and in many instances severe rapidly developing hypotension which might have been difficult to treat was probably avoided by a prophylactic dose of one of these drugs In the elderly patient such prophylaxis is probably of particular value

Oxygen Administration—During spinal or epidural block procedures particularly in geriatrics even if there is no evidence of a marked hypotension intermittent or continuous oxygen administration by bag and mask or nasal pharyngeal catheter should be instituted during the operation and in the immediate postoperative period Although a marked hypotension may not be evident some degree of stagnant hypoxia probably accompanies most spinal and epidural anesthesias

If such an insidious hypoxia occurs in an elderly patient who has a chronic hypoxia from arteriosclerosis noticeable cerebral damage may result In these instances it is usually a member of the patient s family who one to four weeks following an operation notices that father or mother" is acting somewhat peculiarly Often when such cerebral damage develops it is not called to the attention of the surgeon or anesthesiologist and even if it is these physicians may not realize that cerebral damage has occurred or what produced the damage

Starting Intravenous Fluids—The starting of intravenous fluids prior to block procedures likely to produce hypotension is a good prophylactic policy Often additional help is not available to assist the anesthesiologist when the patient develops hypotension A plastic needle is best for such a procedure because it has less tendency to be dislodged from the vein during the positioning of the patient for the anesthetic or the surgical procedure (see Figure 35 page 146)

Avoid the Use of High Spinal and Epidural Blocks in the Patient with a Poor Physical Status—Regional block procedures such as local infiltration field block or peripheral nerve blocking may be the anesthesia of choice in patients of poor physical status—for example those having (1) diabetes with or without arteriosclerotic heart disease (2) hypertension (3) coronary artery disease (4) cerebrovascular diseases and (5) decom

preanesthetic blood pressure was 200 mm systolic and 130 mm diastolic, should cause the physician great concern if the pressure falls to 110 mm systolic and 80 mm diastolic after a spinal or epidural block procedure Such a blood pressure for that individual might be at shock level and might invite coronary occlusion etc Every effort to raise the pressure and assure tissue oxygenation of such a patient must be made

Pulse Rate—The pulse rate becomes rapid and as hypotension becomes more pronounced the rate may be too high to be determinable If corrective therapy is not instituted the pulse becomes imperceptible and cardiac failure and death may ensue

Oxygen Want—When hypotension without marked paralysis of the muscles of respiration is present (a situation which is often seen when spinal or epidural block does not rise above T_6), *the oxygen want is caused by stagnant hypoxia* Slowed circulation of the blood due to vasodilatation is responsible for this hypoxia In the author's opinion hypoxia is not the cause but a result of hypotension in this situation As the anesthesia progresses above the level of T_6 a substantial number of the intercostal nerves are paralyzed and whether or not diaphragmatic action is weakened or ceases tidal hypoxia now coexists with stagnant hypoxia

Early signs and symptoms of oxygen want are yawning nausea vomiting apprehension fear restlessness dizziness tinnitus and headache They are referable to tissue hypoxia of the central nervous system due to the diminished blood pressure The late signs and symptoms of oxygen want are those of depression of the central nervous system as well as of the heart and peripheral vascular system The depression is reflected by drowsiness disorientation coma respiratory arrest shock and death (see Chapter 17 page 160)

Appearance of the Patient—*Cyanosis is usually not seen* when severe hypotension develops during a spinal or epidural block because as shock develops marked capillary constriction in the unanesthetized area usually occurs to compensate for the vasodilatation in the paralyzed area Even though the amount of circulating reduced hemoglobin is markedly increased (5 gm or more per 100 cc of blood) it does not enter the skin capillaries of the upper part of the body and therefore *cyanosis is usually not apparent*

Shock and Death—If therapy to combat the hypotension is not instituted immediately and hypotension with associated hypoxia is allowed to progress the blood pressure becomes unobtainable the pulse is weak slow or unobtainable the skin becomes cold may or may not sweat, and a cadaveric appearance cardiac failure and death ensue

PROPHYLAXIS

The prophylaxis of hypotension consists of

Taking the Blood Pressure—Prior to a regional block procedure particularly a spinal epidural or celiac plexus block a blood pressure cuff should be placed securely on the patient's arm (see Figure 34 page 145) Correct placement of the cuff is essential so that on completion of these blocks time will not be lost in reapplying it

The blood pressure must be checked before doing the block while executing the block (if help for this purpose is available) immediately following the block, and thereafter at frequent intervals

Preblock Administration of Vasoconstrictor Drugs—It has been the policy of the author to give 20 to 50 mg of ephedrine sulfate or 7 5 to 10 mg of Vasoxyl intramuscularly 15 to 20 minutes prior to regional block procedures such as spinal block epidural block and splanchnic (celiac plexus) block which are often accompanied by falls in blood pressure Doses of other vasoconstrictor drugs such as 2 to 5 mg of Neosynephrine 15 to 20 mg of Methedrine 50 to 100 mg of Oenethyl or 15 to 30 mg of Wyamine may be equally effective when given intramuscularly as a prophylactic dose Such doses usually do not alarmingly increase the blood pressure of the hypertensive patient about to receive a spinal or epidural block

Nevertheless objection to the use of a vaso

administration of oxygen is started first depends on the circumstances, but if possible they should be instituted concomitantly.

Administration of Vasoconstrictor Drugs.—If the blood pressure has not stopped its decline and started to rise toward its preoperative level by the time the above therapy has been instituted, it is mandatory that vasoconstrictor drugs be given *intravenously* in an attempt to raise the blood pressure. When used prophylactically the vasoconstrictor drugs may be given intramuscularly, but when hypotension is present peripheral circulation is slowed and drugs given subcutaneously or intramuscularly are *slowly* absorbed. Therefore, *intravenous administration* is the only rational method of giving a vasoconstrictor drug to correct a severe hypotension rapidly. The author agrees with Collins[illegible] that a vasoconstrictor drug is the "keystone" to therapy, and it behooves the physician to choose one or two of these drugs and become thoroughly familiar with their mechanism of action as well as their usual effect.

All vasoconstrictor drugs do not act in the same way. The vasoconstrictor action may depend on: (1) direct action on the arterioles, (2) myocardial stimulation with increased cardiac output, (3) stimulation of the vasomotor center, and/or (4) constriction of the veins. The appropriate vasoconstrictor agent must be chosen and the one selected depends to some degree on whether a fast or slow pulse is noted after a local anesthetic procedure. A slow pulse in the presence of hypotension reflects depression of the myocardium as well as peripheral vasodilatation and therefore would indicate the use of a drug like ephedrine sulfate, which in addition to a peripheral vasoconstrictor action stimulates the heart directly. A fast pulse in the presence of a low blood pressure, on the other hand, would probably indicate increased activity of the heart along with peripheral vasodilatation and suggest the use of a drug which slows the heart rate while producing peripheral constriction of the blood vessels, e.g., Neosynephrine. The more commonly used vasoconstrictors, their dosages, and the fashion in which they act are listed below.

EPHEDRINE SULFATE

Dosage.—15 to 25 mg. *intravenously* repeated at 2 to 5 minute intervals until the preblock blood level is approximated, then the dosage is reduced to 10 mg. and given at intervals necessary to maintain the pressure until it is stabilized.

Action.—It constricts the arterioles, stimulates the cerebral cortex, and increases the cardiac output.[illegible, 29] The pulse rate increases.[illegible, illegible] Ephedrine (and also Arterenol and methamphetamine), according to Cotten *et al.*,[illegible] produce an actual increase in heart force and do not act on the same basis as Neosynephrine or Vasoxyl (see below).

NEOSYNEPHRINE (phenylephrine, Neophryn)

Dosage.—1 to 2 mg. intravenously when given intermittently. However, it is a powerful drug and in the author's opinion is best given by the drip method. The drip solution is prepared by adding 1 cc. of a 1% (10 mg./cc.) Neosynephrine solution to 1000 cc. of intravenous fluid, usually 5% dextrose in distilled water. Initially this mixture is dripped rapidly (100 to 180 drops per minute) until the blood pressure approaches the pre-block level and then it is slowed to a maintenance drip (40 or fewer drops per minute) until the pressure stabilizes. This method of correcting hypotension is the choice of the author because it gives minute to minute control and may be continued in the postoperative period with marked safety until the regional block has dissipated itself.

Action.—It raises the blood pressure by peripheral vasoconstriction and cardiac stimulation.[29, 31] In addition, a bradycardia from compensatory reflexes or depression of the sino-auricular node, an increase in the heart size, and increased stroke volume result.[32] Untoward reactions such as apprehension, excitement and headache are negligible.

VASOXYL (methoxamine hydrochloride)

Dosage.—2 to 3 mg. intravenously accompanied by 7.5 to 10 mg. intramuscularly to provide for a prolonged action.

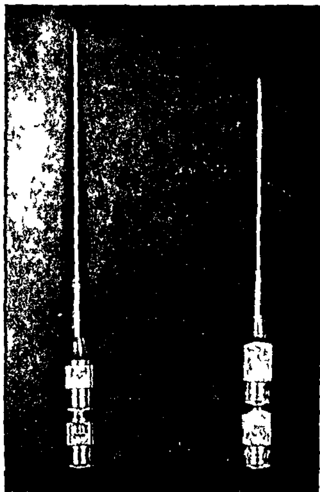


Figure 35 Plastic needles used for intravenous therapy

pensated heart disease Low spinal block or low segmental epidural block i e analgesia below the umbilicus (T_{10}) may be used with relative safety in these patients provided equipment to treat minor degrees of hypotension and oxygen want is present However in patients with any of these problems it may be wise to avoid a high spinal block a high segmental epidural block or splanchnic (celiac *plexus*) block

TREATMENT

Some of the early signs and symptoms of hypotension may resemble a systemic toxic reaction to a vasoconstrictor drug or a psychomotor reaction Therefore a definite diagnosis must be established before instituting routine therapy on the basis of a patient's prodromal signs and symptoms Usually a check of the blood pressure, pulse respirations and the level of analgesia when a spinal or epidural block has been given will give a good clue to the type of reaction which has occurred When hypotension exists early treatment is essential to avoid progression to possible irreversible shock cardiac failure and death In general the treatment is aimed at increasing the oxygen tension of the partially unsaturated venous and arterial blood and increasing the venous return to the heart to maintain cardiac output

Administer Oxygen —One hundred per cent oxygen should be administered immediately, preferably by bag and mask after a clear airway has been established (see Chapter 17 page 163) If respirations are decreased they should be supplemented by manual pressure on the breathing bag or, if such equipment is not available by other forms of artificial respiration Oxygen administration helps to combat both tidal and stagnant hypoxia

Often when the level of anesthesia remains below T_6 the administration of oxygen alone will temporarily fulfill the oxygen needs relieve nausea and vomiting and allow time for the body's compensatory mechanism to correct the hypotension However an intravenous drip should be started simultaneously *with the oxygen administration and if the* hypotension has not reversed by the time the intravenous fluids are started then vasoconstrictor agents should be given through the intravenous tubing

Give Intravenous Fluids —Before or immediately following most regional block procedures particularly spinal and epidural blocks an intravenous drip of 5% dextrose in distilled water or some other suitable fluid should be started so that if hypotension occurs with peripheral vascular collapse time will not be wasted in trying to cannulize a collapsed vein or doing a cutdown However if severe hypotension *develops before the prophylactic* intravenous drip has been started oxygen should be administered either by an assistant or by having the *patient hold* the mask while the intravenous drip is being started Whether the intravenous drip or the

administration of oxygen is started first depends on the circumstances but if possible, they should be instituted concomitantly

Administration of Vasoconstrictor Drugs.—If the blood pressure has not stopped its decline and started to rise toward its preoperative level by the time the above therapy has been instituted it is mandatory that vasoconstrictor drugs be given *intravenously* in an attempt to raise the blood pressure When used prophylactically the vasoconstrictor drugs may be given intramuscularly but when hypotension is present *peripheral circulation is slowed* and drugs given subcutaneously or intramuscularly are *slowly* absorbed Therefore *intravenous administration* is the only rational method of giving a vasoconstrictor drug to correct a severe hypotension rapidly The author agrees with Collins[24] that a vasoconstrictor drug is the "keystone" to therapy and it behooves the physician to choose one or two of these drugs and become thoroughly familiar with their mechanism of action as well as their usual effect

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VASOXYL (methoxamine hydrochloride)

Dosage.—2 to 3 mg intravenously accompanied by 7 5 to 10 mg intramuscularly to provide for a prolonged action

Action—Given intramuscularly, the action of this drug lasts two hours or more[33] It raises the blood pressure by direct peripheral vascular constriction Bradycardia ascribed to reflex inhibition arising from pressure receptors of the carotid sinus and aortic arch occurs[34, 3] Atropine blocks this action which suggests that it is mediated over the vagus It is free of central stimulation effects such as nausea tremor nervousness headache etc[34, 35, 37] However it increases the central venous pressure[36] The small increases in heart force produced by Neosynephrine and Vasoxyl are considered by some to be the result of cardiac dilatation secondary to hypertension and not a result of direct action of these drugs on the myocardium[28]

Vasoxyl in our experience has proven to be a valuable drug both for preventing and for correcting the hypotension following spinal and epidural block

METHEDRINE (d desoxyephedrine, Desoxyn Pervitin Norodin, methamphetamine)

Dosage—5 to 10 mg intravenously followed by 15 to 20 mg intramuscularly

Action—It stimulates the central nervous system constricts peripheral blood vessels and increases cardiac output[38, 40] The duration of action following intramuscular injection is about two hours[38]

OENETHYL (2 methyl amino heptane)

Dosage—5 to 10 mg intravenously repeated at one half minute intervals The pressure is usually elevated to the preblock level in 1 to 2 minutes and this effect lasts 1 to 2 hours[41, 43]

Action—The pressor action is due to arteriolar constriction and perhaps to some action on the veins since this drug also restores venous pressure[41, 43] Tachycardia or bradycardia has not been noted Dizziness occurs in 10 percent of the patients and nausea in 5 percent[24]

WYAMINE (mephentermine sulfate)

Dosage—15 to 20 mg is slowly given intravenously over a one to two minute period The response is prompt lasts 15 to 45 minutes and the injection may be repeated as often as necessary without undesirable side reactions[44]

Action—It has no direct effect on the myocardium[4, 46] The cardiac output remains relatively constant as the blood pressure rises[46, 4] This would indicate that the blood pressure response is due to constriction of the peripheral vessels

LEVOPHED (norepinephrine noradrenalin l arterenol)

Dosage—4 to 8 mg in 1000 cc of 5% dextrose in distilled water administered by the intravenous drip technique

Action—It is the most powerful of all the peripheral vasoconstrictor drugs and has little or no action on the myocardium It causes general vasoconstriction of the arterial capillary and venous blood vessels with a resultant increase in total peripheral resistance[48] It should be used in hypotension following regional block only when other vasoconstrictor agents fail and if it is administered an arm vein of the cubital fossa should be used This is advisable because serious tissue sloughs following the prolonged use of the drug have been reported (see page 150)

ADRENALIN (epinephrine) and PITRESSIN

In general Adrenalin (epinephrine) is not commonly employed because it produces tachycardia anxiety or cardiac irregularities and if tissue hypoxia particularly of the myocardium is present ventricular fibrillation may result If it is used in an emergency when other vasopressor drugs are not available the intravenous Adrenalin drip technique (1 250 000) as popularized by Evans[50] is the preferable method of administration

The untoward side effects of Pitressin too pose a barrier to its use

Head Lowering—Amer[53] believes that the head down position is one of the most effective measures to combat circulatory insufficiency during spinal anesthesia He states

"The mechanism is as follows the altered position causes a displacement of the blood mass from the dilated peripheral vascular areas to the heart and chiefly to the lungs the normal blood volume in the lungs being restored The increase of the pulmonary blood volume results in higher stroke volume and cardiac output and rising blood pressure in the peripheral circulation Clinically the favorable effect of the head down position is clearly perceptible in the form of a rise in the blood pressure and this alteration of the patient's position is often the only measure necessary in slight falls of the blood pressure"

After studying 11 cases with spinal analgesia who were placed in a head down position of 12 degrees Cole [4] concludes It may not be unreasonable to draw the following conclusions from this study (1) Tilting a supine patient so as to place him in the head down position causes a rise in the arterial pressure in the arm and a fall in the pressure in the leg these are probably indicative of an increased blood pressure in the upper half of the body and a decreased pressure in the lower half of the body (2) When the table is tilted the term 'blood pressure loses its meaning and acquires a new significance it is an expression of conditions only in the area being tested (3) The restorative action of head lowering is due in part at least to an actual, significant rapid demonstrable and consistent increase in the blood pressure in the upper extremity and undoubtedly in the brain" He also points out that while the position favors an increased cerebral blood flow because the brain is lower than the heart it is also more difficult for the blood to return "uphill" to the heart

An evaluation of the above statements of Arner [53] and Cole [4] in the light of our own experience would seem to indicate that while head lowering may increase the apparent blood pressure and result in an increased blood flow to the brain it is not a real substitute for other therapy detailed above As a temporary restorative method without other treatment it may result in a precarious situation when the head down position is resolved at the end of surgery particularly if 500 cc or more of blood is lost Should the head down position (Fowler's position) be maintained in the postoperative period to prevent hypotension by draining the blood from the lower part of the body particularly the legs the collapsed blood vessels in these areas predispose to thrombosis and its subsequent complications

Correcting Complications of the Oxygen Want Which Are Secondary to the Hypotension—*Convulsions*—When convulsions occur from oxygen want they are usually easily corrected by oxygen administration Short acting intravenous barbiturates are in most instances not indicated and if given may actually be detrimental They should be given only if the convulsions are not brought under control after three to five minutes of effective oxygen administration If such a situation develops then small doses of intravenous pentothal (50 to 75 mg) may be administered at two to five minute intervals until the convulsions cease

Cardiac Failure—If cardiac failure occurs during a bout of oxygen want resuscitation of the heart, as described in Chapter 7 page 74 must be instituted

Posthypoxia Cerebral Edema—Following periods of oxygen want, cerebral edema may persist and this should not be ignored Its signs and symptoms should be recognized and treatment instituted as described in Chapter 7 page 80

Availability of Additional Help—When severe hypotension occurs one physician should assume charge of the situation and give directions in an orderly fashion to avoid confusion In most cases he should remain at the head of the table maintaining the airway and preventing oxygen want The starting of intravenous fluids the preparation of vasoconstrictor drugs and their administration should be under his direction but should be performed by his assistants However if they are unable to start the intravenous fluid quickly and with dexterity, he must then do the venipuncture himself

If cardiac failure occurs it may be neces



Figure 36 Right lower extremity showing ulceration and necrosis of tissue following intravenous infusion of arterenol (Levophed) into an ankle vein (A) Lies along course of the long saphenous vein and (B) a more detailed view of area of ulceration about the knee (contributed by Unicchio *et al*)

sary for him to delegate the artificial *respira*tion to someone else and perform the manual systole However, if this last mentioned complication occurs when both an anesthesiologist and surgeon are present each should remain at his *position* the anesthesiologist making an attempt to raise the blood pressure and maintain respirations and the surgeon doing the manual systole (see Chapter 7 page 75)

COMMENTS

Use of Vasoconstrictor Drugs in Patients with Cardiovascular Diseases—While vasoconstrictor agents are generally contraindicated by cardiovascular disease particularly in severe hypertension it is to be noted that the hypertensive patient may suffer a greater and more serious fall in blood pressure than the one with a normal pressure when under spinal anesthesia Hence, a pressor drug properly used may be especially valuable prophylactically as well as for corrective therapy

If vasoconstrictor drugs are not used to correct hypotension particularly in patients with myocardial damage coronary disease arteriosclerosis etc a complication due to low blood pressure, such as thrombosis may occur

Vasoconstrictor Drugs Not Indicated in Hypotension from Hemorrhage—When hypotension occurs during a surgical procedure in which regional block is being used the *amount of blood loss should always be deter*mined If blood loss is great vasoconstrictor drugs may be used to support the pressure for a short interval but they are no substitute for replacement therapy with blood plasma or *volume expanders In this type of circum*stance the administration of blood solves the problem most satisfactorily

Carefully Select Drug and Site for Prolonged Intravenous Infusions of Vasoconstrictor Drugs—When it is necessary to use a continuous intravenous infusion of a vasoconstrictor drug to raise and maintain the blood pressure drugs and sites of injection must not be chosen at random because of the danger of necrosis

Neosynephrine is the author s choice for raising the blood pressure when a continuous intravenous technique is indicated because it raises the blood pressure by peripheral vaso constriction as well as cardiac stimulation[23] *Cardiac stimulation is often indicated in these* cases because most local anesthetic agents are known to depress the myocardium In addition Neosynephrine infusions containing 10 mg of Neosynephrine per 1 000 cc of fluid may be started in either the leg or arm veins in the average patient even if a moderate degree of arteriosclerosis and intermittent claudication is present without danger of necrosis and slough at the site of injection

On the other hand if the patient s pressure does not respond to *Neosynephrine or the* cardiac status precludes the use of a vasoconstrictor drug that stimulates the heart a more powerful peripheral vasoconstrictor with little

or no cardiac action such as Levophed (levarterenol norepinephrine) may be indicated. If so the site of infusion must be carefully selected and extravasation into the tissues guarded against. Patients who have diabetes, arteriosclerosis with or without intermittent claudication, cardiac decompensation, a recent coronary occlusion or a low blood pressure may develop a slough at the site of infusion or along the course of the infused vein should the Levophed solution be started in the leg.[7, 48, 51] Urecchio, Calenda and Cutts[5] report a case with severe intermittent claudication who was given 1,000 cc. of Levophed (4 mg. in 1,000 cc.) at the rate of 20 drops per minute and who developed such severe gangrene of the superficial and subcutaneous tissue that a right midthigh amputation was necessary (see Figure 36, page 150). As a result they advise that "... in the presence of occlusive vascular diseases this drug should be used with extreme caution, if at all, and should be given in one of the arms." Kurland and Malach[56] report one case of slough following the use of continuous Levophed drip in vein of the ankle, and Greenwald *et al.*[7] report two. The author has seen one case of gangrene of the finger, one slough of the skin of the ankle and one near slough of the skin of the ankle following the use of Levophed (see Figure 37, page 151; Figure 38, page 152; and Figure 39, page 152).

From experience at the Mason Clinic with Levophed as well as study of the cases cited above, the author recommends that, regardless of the patient's physical status, the veins in the cubital fossa be used when powerful vasoconstrictor solutions such as Levophed are being infused because the collateral circulation is greater in the arm than in the leg.[59] Nevertheless, even if the arm is used, an occasional case may develop an area of gangrene in the hand or other part of the upper extremity if the patient has arteriosclerosis (Figure 37, page 150). It is also interesting to note that deAlvarez[60] has seen a case in which Levophed was started in one of the upper extremities and an area of gangrene developed in the palm of the other. These two cases would indicate that any area of the body where there is decreased circulation may be susceptible to the effects of Levophed regardless of where the infusion is started.

Should extravasation of Levophed solutions occur at the site of the infusion or should any area of the body, particularly all or part of the extremity in which the infusion is running, show evidence of impaired circulation, treatment as described in Chapter 10, page 107, must be instituted immediately.

Degree of Hypotension from Spinal (Subarachnoid) Block and from Spinal Epidural Block.—Some authors feel that spinal epidural block alters the blood pressure very little even in the presence of a high sensory

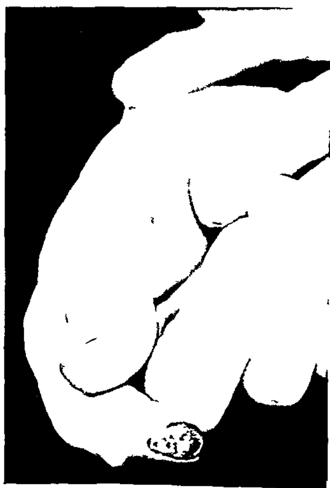


Figure 37. Gangrene of the tip of the index finger of a 50 year old diabetic patient following the intravenous drip administration of Levophed in the cubital fossa during diabetic coma and shock. *Note.*—Most diabetics past the age of 40 have associated arteriosclerosis.

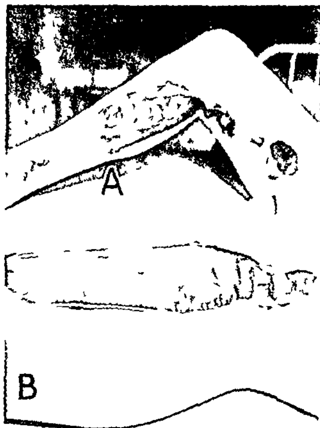


Figure 36 Right lower extremity showing ulceration and necrosis of tissue following intravenous infusion of arterenol (Levophed) into an ankle vein (A) Lies along course of the long saphenous vein and (B) a more detailed view of area of ulceration about the knee (contributed by Unechio *et al* [4])

sary for him to delegate the artificial respiration to someone else and perform the manual systole However if this last mentioned complication occurs when both an anesthesiologist and surgeon are present each should remain at his position the anesthesiologist making an attempt to raise the blood pressure and maintain respirations and the *surgeon doing the* manual systole (*see* Chapter 7 page 73)

COMMENTS

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On the other hand if the patients pressure does not *respond to* Neosynephrine or the cardiac status precludes the use of a vasoconstrictor drug that stimulates the heart a more powerful peripheral vasoconstrictor with little

respiration as well as any intra abdominal manipulation may further predispose to hypotension Therefore while this block is by definition a peripheral nerve block and its discussion should perhaps be included in Part I it is included in this chapter because the etiology and treatment of the hypotension is similar to that of spinal and epidural block As noted hypotension from this block usually does not result unless positional changes or surgical manipulations occur

Positional Changes—Usually when splanchnic block is performed for diseases of the pancreas (pancreatitis etc) the blood pressure will not change if the patient remains in the supine position However if he sits up or attempts to get out of bed he will feel faint or actually faint because a rapid hypotension will develop This may be avoided following a block with a local anesthetic agent by restricting the patient to bed for the physiologic duration of the drug or by the use of a tight abdominal binder and elastic stockings The use of the latter is essential when an alcohol or phenol splanchnic block for the relief of intractable cancer pain is performed if the patient is to be ambulatory within a few hours after the block In most instances these devices must be applied for one or two weeks until the patient s compensatory mechanisms adjust Even if the above noted precautions are taken someone should be in attendance when the patient first attempts to assume the vertical position otherwise he may faint fall and sustain an injury

Surgical Manipulations—When the block is combined with an intercostal block for upper abdominal surgery in the adult often a drop in blood pressure does not occur until surgery is started In over 1 450 patients anesthetized by the author and his associates with intercostal deep splanchnic (celiac plexus) block for upper abdominal surgery it was noted that 80% did not show a fall in blood pressure until the abdomen was opened or manipulation of the abdominal viscera began When the abdomen was closed in these patients it was not uncommon for the pressure to resume its presurgical level We have observed that such a fall in blood pressure can be adequately controlled during and after surgery by either prophylactic intramuscular dosages of the vasoconstrictor drugs or a Neo synephrine drip

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Figure 38 Slough of the skin of the ankle at the site of administration of Levophed to an elderly patient who suffered shock following a cerebrovascular accident No extravasation of Levophed had occurred during the infusion



block.[61, 0] Others have found that blood pressure descents during epidural anesthesia are comparable to those found following spinal anesthesia and our observation of over 1700 cases in which spinal epidural blocks have been executed would substantiate the findings of this second group of physicians.[71, 7] Abajian[7] states "There is sometimes a fall in arterial pressure with peridural segmental anesthesia even sometimes to the same degree as in subarachnoid block and not merely 20 to 30 mm of mercury as proclaimed by previous publications on epidural anesthesia."

Hypotension Following Splanchnic (Celiac Plexus) Block.—When splanchnic block is used alone for a diagnostic or therapeutic block hypotension usually does not occur unless the patient is moved from a horizontal to a vertical position. If it is used together with a bilateral block of the lower seven or eight intercostal nerves for upper abdominal surgery paralysis of some of the muscles of



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Figure 39 Blistering following the use of Levophed in an ankle infusion No extravasation of Levophed had occurred during the infusion

respiration as well as any intra abdominal manipulation may further predispose to hypotension Therefore while this block is by definition a peripheral nerve block and its discussion should perhaps be included in Part I it is included in this chapter because the etiology and treatment of the hypotension is similar to that of spinal and epidural block As noted hypotension from this block usually does not result unless positional changes or surgical manipulations occur

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and the physiological effects of these drugs may be dissipated soon after the patient returns to his room The removal of these supports of the peripheral vascular system prior to the dissipation of the effects of the local anesthetic drug may result in severe hypotension

Discontinuation of Intravenous Administration of Fluids.—Usually prior to or during major surgical procedures intravenous fluids are started which may temporarily compensate for a relative decrease in blood volume caused by vasodilatation from a regional block or an actual decrease in blood volume caused by hemorrhage When the patient is returned to his room the intravenous infusion may be purposely discontinued or the intravenous needle may come out of the vein and may not be replaced either because the physician feels that ample fluid has been given or because no one is available to restart it If the body excretes or metabolizes the previously administered fluids before the physiological effects of the local anesthetic drug have disappeared, hypotension may result

Rapid Changes in Position.—When vasodilatation following the paralysis of portions of the sympathetic nervous system occurs and the patient is placed on the operating table in the desired position the body may compensate adequately and maintain the blood pressure If however after surgery the patient is moved rapidly and roughly from the operating table to the stretcher or from the stretcher to the bed a severe hypotension which may or may not respond to treatment may result[3] In some instances rapid changing of position has resulted in death[3] Hypotension from rapid changes in position has been noted with general as well as regional anesthesia but it seems to occur more readily after regional block procedures which paralyze a large portion of the nervous system

Soffer and Sweet[3] state in their summary "Significant gravitational movement of blood in the legs occurs following minor position shifts during the state of vasomotor paralysis produced by certain autonomic blocking drugs high or low spinal anesthesia, and to a lesser extent during thiopental sodium [Pentothal] anesthesia Depression or elevation of the lower extremities may therefore be utilized to lower or raise the subject's blood pressure as the situation demands One must however, be constantly aware of the drastic circulatory changes that may rapidly ensue if these position shifts are produced or resolved suddenly and without continuous observation Three cases are described that illustrate the blood pressure changes that may occur during spinal anesthesia as a result of the assumption or resolution of the lithotomy position The fatal outcome of case 3 was directly related to such a postural shift of the lower extremities"

SIGNS AND SYMPTOMS

The signs and symptoms of hypotension from a spinal block or an epidural block are the same regardless of when they occur (see Chapter 15 page 143)

PROPHYLAXIS

To prevent hypotension from developing in the immediate postoperative period the following precautions should be observed

Discontinue Oxygen Administration via the Anesthetic Machine During the Operation.—One half to three fourths of an hour before the end of surgery the patient should be allowed to breathe room air and should be carefully observed to see if his blood pressure drops The purpose of disconnecting the anesthetic machine from the patient is to evaluate the extent to which carbon dioxide accumulation may have helped maintain the blood pressure If hypotension develops after such a test appropriate steps to correct it and maintain adequate blood pressure in the postoperative period may be formulated

Watch Patient Carefully When Vasoconstrictor Drug Therapy Is Suspended.—If a patient has been receiving a vasoconstrictor drug intravenously or intramuscularly to maintain his pressure during surgery this type of therapy should not be suddenly withdrawn

CHAPTER 16

Hypotension from Spinal and Epidural Block in the Immediate Postoperative Period

IF PROPHYLACTIC measures are not taken prior to returning the patient to his room hypotension in the immediate postoperative period may occur following high spinal epidural blocks and intercostal splanchnic (celiac plexus) blocks but it occurs infrequently following other types of regional block

ETIOLOGY

The main reason for postoperative hypotension following such blocks as spinal etc is paralysis of the nerves in the blocked area with consequent vasodilatation and pooling of blood in this anesthetized area During surgery this hypotension may be compensated for by administering oxygen vasoconstrictor drugs minimizing changes in position of the patient and replacement therapy i e intravenous infusions and blood transfusions However at the completion of surgery the following often take place (1) the oxygen via the anesthetic machine is stopped (2) the intravenous administration of the vasoconstrictor drug is discontinued or a previous intramuscular dose of this type of drug has dissipated itself (3) the intravenous fluid therapy is discontinued and (4) sudden changes in position occur such as moving the patient from the operating table Any one of these factors or a combination of more than one may result in hypotension in the immediate postoperative period

Discontinuation of Oxygen via an Anesthetic Machine —When the intercostal nerves and a large part of the sympathetic nervous system are paralyzed by a regional block tidal hypoxia from the decreased respiratory function and stagnant hypoxia from the pooling of blood result To compensate for this the patient is often given oxygen via the anesthetic machine And, in many instances even though the regional anesthesia is satisfactory Pentothal (thiobarbiturate) anesthesia may be given when the procedure is prolonged or the patient becomes restless Both the administration of oxygen via the machine and the Pentothal narcosis unless carefully managed tend to result in accumulation of carbon dioxide in the alveoli and thus an abnormally high blood level of carbon dioxide although the oxygen saturation of the blood may be within normal limits This carbon dioxide accumulation may partially maintain or raise the blood pressure At the end of surgery when the machine is disconnected from the patient this excess of CO_2 may be eliminated and with the supporting effect of the carbon dioxide removed hypotension may result The mechanism of this type of low blood pressure is probably the same as that responsible for cyclopropane shock [1, 4]

Discontinuation of Vasoconstrictor Drug Therapy—Often a Neosynephrine (phenylephrine) or Levophed (norepinephrine) drip which has been running during the surgical procedure to maintain the blood pressure will be discontinued at the end of surgery for fear that it may run too fast in the immediate postoperative period Vasoconstrictor drugs are also often given intramuscularly during the operation to prevent falls in blood pressure

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Oxygen Want Secondary to the Hypotension Following Spinal or Epidural Block

NOT INFREQUENTLY respiratory embarrassment (hypoxia) and occasionally respiratory arrest (anoxia) occur when a purposely administered spinal or epidural block procedure is executed but seldom occur during local infiltration or peripheral nerve block.

In the author's opinion oxygen want during spinal and epidural block procedures is secondary to hypotension. It must be emphasized that the hypotension precedes the oxygen want although as the anesthesia rises and paralyzes the intercostal and phrenic nerves the resultant decrease in tidal exchange exerts an additive effect. Since hypotension and oxygen want may coexist the physician must be cognizant of the cause and treatment of both so that they may be treated simultaneously therefore this chapter and the preceding two should be considered together.

Before examining oxygen want from spinal and epidural nerve blocks the seven types of hypoxia as defined in Table X page 42 and the difference between the types of oxygen want which may occur in regional block procedures as shown in Table XI page 46 must be clearly understood (see Chapter 4, page 42).

ETIOLOGY

In general hypoxia or anoxia following a spinal or epidural block is primarily the result of hypotension with or without paralysis of the nerves controlling the muscles of respiration. Other factors which may occasionally result in a hypoxia during these block procedures are (1) a systemic toxic reaction to a vasoconstrictor drug administered prophylactically, (2) a systemic toxic reaction to the local anesthetic drug (3) injudicious premedication or (4) a combination of these. It must also be remembered that these regional blocks are often selected for patients with a poor physical status i.e. those suffering from cardiac disease pulmonary disease thyroid diseases etc. and that such a disease may have produced chronic hypoxia prior to the induction of the anesthetic. Therefore pre-existing diseases which produce oxygen want may be termed "contributing factors" in that they accentuate the hypoxia which may result from a regional nerve block procedure.

Hypotension With or Without Paralysis of the Nerves Controlling the Muscles of Respiration Following Spinal or Epidural Blocks.—Hypoxia from a spinal or epidural block may be due to hypotension from vascular dilatation and pooling of blood (stagnant hypoxia) to a paralysis of the intercostal muscles and diaphragm (tidal hypoxia) or to a combination of these. While hypotension is the initial and principal cause in the author's opinion the statement of Seevers and Waters on page 139 must be evaluated. The direct depressing effect on the respiratory and cardiovascular centers in the medulla must also be considered when a high or total spinal anesthesia follows the inadvertent subarachnoid injection of too massive a volume or too high a concentration of a local anesthetic

when he is returned to his room Whenever vasoconstrictor drug therapy is withdrawn and particularly if the effects of the local anesthetic drug have not worn off the patient must be carefully observed Here again when a drip of a vasoconstrictor drug is being given during surgery, it is best to discontinue the drip a half to three quarters of an hour prior to the end of surgery as a test Then if blood pressure falls measures to support it in the postoperative period may be undertaken

Do Not Discontinue Intravenous Fluid Therapy Without Careful Observation of the Patient—Normal saline solutions 5% dextrose in distilled water and other physiological solutions may temporarily increase the blood volume and sustain blood pressure if run rapidly When fluids are suddenly discontinued the patient's pressure may drop especially while the anesthetic remains effective Therefore when fluid therapy is discontinued the patient *must be carefully observed for at least* one to two hours

Move Patient Carefully—Under no circumstances should patients who have had anesthesia be moved rapidly Those patients who are in serious condition following surgery should be moved directly from the operating table to the bed in order to reduce the number of times they must be moved Jerking and pushing a patient is not to be tolerated and adequate help should be available when positioning and transporting a patient

TREATMENT

The treatment of hypotension is the same regardless of why it occurs (see Chapter 15 page 146) *However, when hypotension develops in the postoperative period the possibility that hemorrhage is causing it must be particularly kept in mind*

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weakness, delirium and coma. The vomiting center is stimulated by the hypoxia, so nausea is an early symptom followed later by retching and vomiting. Muscular incoordination and twitching are relatively early signs of hypoxia which progress to convulsions as severe hypoxia develops, but muscular relaxation usually occurs prior to death, probably because of central nervous system depression.

It must be remembered that heavy premedication will often hide or mask these signs and symptoms, and this, in addition to the fact that cyanosis may not occur to indicate oxygen want (see page 162), accounts for the fact that if a patient is not carefully attended during regional block analgesia—particularly a spinal block or an epidural block—death may occur from hypotension and hypoxia without any warning signs or symptoms. Instances have occurred where the surgeon or obstetrician acting as his own anesthesiologist has given what he thought was a "low" spinal, left the patient unattended, went to scrub, returned, gowned and gloved himself, draped the patient only to find when making the incision that the patient did not bleed and was dead.

Respiratory Manifestations.—There is usually no change in the respiratory rate or depth when hypoxia starts to develop from hypotension, but as it becomes more severe tachypnea occurs. If oxygen want is allowed to progress, the tachypnea is usually followed by a depression in rate and depth of respiration and thence by cessation. Dyspnea, a subjective sensation, may or may not develop. Comroe and Dripps[11] point out that the degree of dyspnea varies with the individual and they state "The respiratory minute volume at which dyspnea appears is about a third to a half of the total volume of air that an individual can breathe at the time by maximal voluntary effort."

On the contrary, if oxygen want develops from overstimulation following the administration of a prophylactic dose of a vasoconstrictor drug, the patient's respirations usually increase in response to the drug.

Cardiovascular Manifestations.—In most instances when a mild degree of oxygen want is produced following spinal and epidural block in a normal and healthy individual, the blood pressure and pulse rate show little or no change. Low blood pressures usually return to normal in a short period of time as the compensatory mechanisms of the body come into play or the drug is detoxified. This mild type of oxygen want often goes unnoticed unless a careful check for it is made. In patients with coronary heart disease, anginal pain may be a symptom of moderate degrees of oxygen insufficiency.

Following a spinal or epidural block, the marked changes in blood pressure and pulse rate response depend on the cause of the oxygen want.

Hypotension from Spinal or Epidural Block.—When severe oxygen want develops following the onset of a high spinal or epidural anesthetic, there is no momentary initial rise in blood pressure and pulse—the blood pressure falls precipitously at first and the pulse rate increases. If at this time adequate corrective measures are not taken and if the patient's compensatory mechanism does not function, cardiac failure and death may ensue.

Note that in spinal and epidural block procedures where moderate or severe hypotension is most often the principal cause of oxygen want, the slow, full and bounding pulse which so often accompanies moderate to severe primary oxygen want is seldom present (see Table XI, page 46).

Systemic Toxic Reactions to Local Anesthetic Drugs.—Normally this type of reaction is not associated with spinal anesthesia because only small amounts of the local anesthetic drugs are employed. It may occur during an epidural block where a large amount of the local anesthetic drug is injected. When a systemic toxic reaction results and its onset is slow, blood pressure and pulse may rise initially, reflecting the stimulatory phase of the reaction, and then fall as the reaction progresses to the depression phase. This initial rise is the opposite of that seen following hypotension from spinal or epidural block. However, if the systemic toxic reaction has a rapid onset or is not recognized until the stimulatory phase of the reaction has passed, the

agent since in this situation a high concentration of the drug reaches the cranial cerebrospinal fluid

Systemic Toxic Reactions to Vasoconstrictor Drugs Administered Prophylactically —*In* these cases the hypoxia is caused by overstimulation of the central nervous system and by an increased metabolic rate of the body both of which are caused by the vasoconstrictor drug It is a demand type of hypoxia and is primarily the result of oxygen want and is not associated with hypotension

Systemic Toxic Reactions to Local Anesthetic Drugs —The hypoxia which results from this type of reaction is usually attributed *to the depression of the respiratory center in* the medulla which follows overstimulation [1, 2] However in light of recently published articles the question arises as to whether or not in some of these cases the hypoxia may not be secondary to myocardial depression and/or peripheral vascular collapse (see Chapter 1 page 16) [3, 9] The hypoxia following this type of reaction could then be classified tidal as well as stagnant or a combination of these

It should be noted that this type of reaction may occasionally follow an epidural block where large volumes of local anesthetic solutions are employed but seldom accompanies spinal anesthesia where relatively small doses of the local anesthetic agents are injected

Premedication —Excessive doses of barbiturates or opiates drugs which depress respiratory depth and rate respectively may result in *tidal* hypoxia Even if they do usually no problem develops unless further hypoxia is produced by a high spinal etc

Contributing Factors —Often regional blocks are given as the anesthesia of choice to the patient with a poor physical status who has a chronic hypoxia from diseases such as tuberculosis (alveolar hypoxia) emphysema (tidal and alveolar hypoxia) anemia (hemoglobic hypoxia) hyperthyroidism (demand hypoxia) or hyperpyrexia (demand hypoxia) It has also been the anesthesia of choice in high altitudes (atmospheric hypoxia) and in patients with drug poisoning (histotoxic hypoxia) because of the difficulties involved in administering inhalation anesthetics under these conditions When administering a regional block in a situation where any one of these problems exists it must be realized that hypoxia from hypotension or paralysis of the muscles of respiration caused by the anesthetic procedure will automatically accentuate tissue hypoxia and may result in obvious respiratory distress If the physician is not cognizant of this possibility and does not take steps to alleviate the hypoxia an occasional patient—especially the geriatric case with advanced arteriosclerosis—may suffer irreversible tissue damage—particularly of the brain Rovenstine [10] has repeatedly emphasized this possibility *Brain damage of this type may* also occur from hypoxia during general anesthesia and is not peculiar to regional block [13]

SIGNS AND SYMPTOMS

The signs and symptoms of oxygen want in regional anesthesia depend on the speed with which the oxygen want develops and the physical status of the patient If the oxygen want develops rapidly the early signs may be absent and those of advanced severity appear immediately The physical status of the patient also influences the time of appearance as well as the degree of hypoxia to a considerable extent The patient with diminished cardiovascular and/or respiratory reserve who is given a spinal or epidural block a procedure which in a healthy normal individual would cause no problem may show the signs of severe hypoxia very early

In general the following signs and symptoms of oxygen want are listed in the order of their usual appearance They may progress from one to the next either slowly or rapidly

Cortical Manifestations —Early in the course of oxygen want the patient may yawn feel overconfident and show impaired judgment and poor mental concentration pugnacity develops vision is impaired the patient may complain of a severe headache and anxiety vertigo and a sense of "air hunger" may follow If the cause is not corrected the hypoxia progresses to severe oxygen want with

weakness, delirium, and coma. The vomiting center is stimulated by the hypoxia; consequently, nausea is an early symptom followed later by retching and vomiting. Muscular incoordination and twitching are relatively early signs of hypoxia which progress to convulsions as severe hypoxia develops, but muscular relaxation usually occurs prior to death, probably because of central nervous system depression.

It must be remembered that heavy premedication will often hide or mask these signs and symptoms, and this, in addition to the fact that cyanosis may not occur to indicate oxygen want (see page 162), accounts for the fact that if a patient is not carefully attended during regional block analgesia—particularly a spinal block or an epidural block—death may occur from hypotension and hypoxia without any warning signs or symptoms. Instances have occurred where the surgeon or obstetrician acting as his own anesthesiologist has given what he thought was a "low" spinal, left the patient unattended, went to scrub, returned, gowned and gloved himself, draped the patient, only to find when making the incision that the patient did not bleed and was dead.

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blood pressure and pulse response may be the same as that seen following hypotension For blood pressure and pulse response comparisons in these different situations see Table XI page 46

Systemic Reaction from the Vasoconstrictor Drug—When vasoconstrictor drugs are administered prophylactically the patient may show signs and symptoms resembling oxygen want from hypotension However in these cases the blood pressure is elevated the pulse rapid full and strong

Appearance of the Patient—Following a spinal block or an epidural block, the appearance of the patient varies with the cause of the oxygen want

Oxygen Want Secondary to Hypotension—In most instances patients with severe oxygen want secondary to hypotension following spinal and epidural block are cadaveric in appearance Cyanosis which is often a reliable sign during general anesthesia local infiltration and peripheral nerve block is usually not present in these instances The reason for this is easily understood The appearance of cyanosis is due to the amount of reduced hemoglobin in the capillary blood of the skin At least 5 grams of reduced hemoglobin per 100 cc of blood must be present before cyanosis of the skin is observable If there is marked peripheral constriction in the unanesthetized area to compensate for the vasodilatation in the blocked area a usual occurrence during hypotension following spinal anesthesia or an epidural block cyanosis of the upper part of the body will not be seen because there is little or no superficial circulation although there may be over 5 grams of reduced hemoglobin per 100 cc of blood

Oxygen Want Secondary to Hypertension—When a systemic reaction to a vasoconstrictor drug ensues the blood pressure is raised metabolism increased and this in turn produces signs of oxygen want Nevertheless the constriction of the peripheral vessels prevents blood from reaching the skin and consequently the patient may appear pale

Primary Oxygen Want from a Systemic Toxic Reaction to a Local Anesthetic Drug—Following a systemic toxic reaction to a local anesthetic drug after an epidural block cyanosis may develop (see Chapter 4, page 45)

PROPHYLAXIS

Prophylaxis of oxygen want following a spinal block or an epidural block is essentially the same as that listed for avoidance of hypotension and consists of *prevention of systemic toxic reactions preblock administration of vasoconstrictor drugs, oxygen administration starting of intravenous fluids and the avoidance of high spinal and high epidural blocks in the patient with a poor physical status* (see Chapter 15 page 144) One should not overlook the fact that when vasoconstrictor drugs are given prophylactically before block oxygen want may develop from a systemic reaction to that drug (see Chapter 3 page 37)

TREATMENT

When severe oxygen want occurs additional personnel is most helpful One physician should assume charge of the situation and give directions in an orderly fashion to avoid confusion In most cases, he should remain at the head of the table maintaining the airway and giving artificial respiration The starting of intravenous fluids the preparation of vasoconstrictor drugs and their administration should be under his direction but should be performed by his assistants If cardiac failure occurs it may be necessary for him to delegate the artificial respiration to someone else and perform the manual systole However if this complication occurs when both an anesthesiologist and surgeon are present each should remain at his position—the anesthesiologist to maintain respirations and the surgeon to perform the manual systole

The treatment of oxygen want following spinal and epidural block depends on the etiology

Oxygen want following spinal and epidural block is most often associated with hypotension but since it may be caused by a sys

tonic toxic reaction to either the local anesthetic or vasoconstrictor drug, a definite diagnosis of its cause must be established before automatically instituting therapy. That the patient has had a spinal or epidural anesthetic does not mean that he necessarily suffers from a resulting hypotension. *Usually a check of the blood pressure, pulse, respirations, degree of aeration of the lungs and the level of analgesia* will give a good clue as to the type of reaction.

Oxygen Want Following Hypotension.—When oxygen want is caused by hypotension the hypotension must be immediately corrected as indicated in Chapter 15, page 146. As emphasized in Chapter 15, the starting of an intravenous drip either prophylactically or as soon as the block is completed is imperative to assure a means of introducing drugs intravenously should peripheral vascular collapse ensue. At the same time that these therapeutic necessities are being accomplished any respiratory embarrassment must be corrected.

Clear the Airway.—If the patient does not lose consciousness, he will in most instances maintain a patent unobstructed airway. However, if the patient becomes unconscious either an oral airway or preferably an endotracheal tube with an inflatable cuff should be inserted. The endotracheal tube with a cuff is emphasized because, following correct placement, the cuff may be inflated; then if vomiting occurs, aspiration with contamination of the respiratory tract will not result. The cuffed endotracheal tube also prevents air from passing down the esophagus and dilating the stomach. Dilatation of the stomach is not an uncommon occurrence if artificial respiration with bag and mask equipment is performed with only an oral airway in place. If this is not observed and treated by the insertion of a Levin tube, acute dilatation of the stomach can itself result in death.

If a semi-conscious patient vomits prior to the insertion of a cuffed tracheal tube, it is likely that aspiration will occur. This necessitates either the placement of an endotracheal tube and blind tracheal suction or bronchoscopy with removal of fluid and food particles from the tracheobronchial tree by direct vision. The latter is preferable because large particles of food can be removed and an adequate tracheobronchial toilet can be performed. The bronchoscope should have a side arm for the insufflation of oxygen during the bronchoscopy. *It behooves the physician performing regional block procedures to have a laryngoscope and bronchoscope available and to know how to use them* (see Chapter 18, page 168).

Immediately following bronchoscopy an endotracheal tube with a cuff should be placed and the cuff inflated. This will prevent further aspiration should vomiting recur. It should also be remembered that the patient who has aspirated is likely to develop a chemical pneumonitis from the acid stomach content. Intensive antibiotic therapy should be given to prevent secondary infection of the damaged lung parenchyma.

Administer Oxygen.—If the patient has not lost consciousness and can maintain his own anatomical airway but has a decreased respiratory rate or an altered rhythm, he should be made to breathe 100% oxygen if this is available. If the respirations are extremely shallow and rapid it may be advisable to aid them by rhythmically squeezing the reservoir bag of the oxygen equipment during the inspiratory phase.

If the patient has lost consciousness and has an obstructed airway, this should be corrected as described above and in Chapter 18, page 167. Then if respirations are decreased or cease because of paralysis of the intercostal nerves, the phrenic nerves and/or depression of the respiratory center, artificial respiration must be instituted immediately and maintained until spontaneous respiration is reestablished. If a machine and/or oxygen are not immediately available (although they should be), artificial respiration may be accomplished by either one of the manual methods, i.e., Silvester method, etc., or mouth to mouth breathing. However, in any case the most satisfactory method of administering artificial respiration is by bag and mask. This may consist of simple pieces of apparatus, i.e.,

blood pressure and pulse response may be the same as that seen following hypotension For blood pressure and pulse response comparisons in these different situations see Table XI page 46

Systemic Reaction from the Vasoconstrictor Drug—When vasoconstrictor drugs are administered prophylactically the patient may show signs and symptoms resembling oxygen want from hypotension However in these cases the blood pressure is elevated the pulse rapid full and strong

Appearance of the Patient—*Following* a spinal block or an epidural block the appearance of the patient varies with the cause of the oxygen want

Oxygen Want Secondary to Hypotension—In most instances patients with severe oxygen want secondary to hypotension following spinal and epidural block are cadaveric in appearance Cyanosis which is often a reliable sign during general anesthesia local infiltration and peripheral nerve block is usually not present in these instances The reason for this is easily understood The appearance of cyanosis is due to the amount of reduced hemoglobin in the capillary blood of the skin At least 5 grams of reduced hemoglobin per 100 cc of blood must be present before cyanosis of the skin is observable If there is marked peripheral constriction in the unanesthetized area to compensate for the vasodilatation in the blocked area a usual occurrence during hypotension following spinal anesthesia or an epidural block cyanosis of the upper part of the body will not be seen because there is little or no superficial circulation although there may be over 5 grams of reduced hemoglobin per 100 cc of blood

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Primary Oxygen Want from a Systemic Toxic Reaction to a Local Anesthetic Drug—Following a systemic toxic reaction to a local anesthetic drug after an epidural block, cyanosis may develop (see Chapter 4 page 45)

PROPHYLAXIS

Prophylaxis of oxygen want following a spinal block or an epidural block is essentially the same as that listed for avoidance of hypotension and consists of *prevention of systemic toxic reactions preblock administration of vasoconstrictor drugs oxygen administration starting of intravenous fluids and the avoidance of high spinal and high epidural blocks in the patient with a poor physical status* (see Chapter 15 page 144) One should not overlook the fact that when vasoconstrictor drugs are given prophylactically before block oxygen want may develop from a systemic reaction to that drug (see Chapter 3 page 37)

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CHAPTER 18

Nausea and Vomiting

NAUSEA or vomiting often accompanies spinal and epidural block but seldom accompanies peripheral nerve block or local infiltration unless a systemic toxic reaction to the local anesthetic drug ensues. When vomiting occurs the outcome may be catastrophic unless the physician has the equipment immediately available to treat the patient and knows how to use it.

ETIOLOGY

Nausea is believed to be caused by stretching of the stomach wall, esophagus or duodenum with tension upon the nerve endings in these structures. Thus it would seem that the stimuli which induce nausea are the same as those which cause visceral pain except that they are lower in intensity.[1]

Vomiting is usually the result of stimulation of the vomiting center in the medulla. Nausea may, if of sufficient magnitude, result in vomiting but there is more than one type of stimulus responsible for vomiting. Best and Taylor[1] state "The mechanism of vomiting involves the coordinated actions of the muscles of the stomach, esophagus, and abdominal wall. The act may also be associated with antiperistaltic movements in the intestine. The muscular mechanisms are governed by a center in the medulla which discharges impulses along numerous efferent nerves, and may be influenced by afferent impulses arising in the stomach, in other viscera or in practically any region of the body. Or the center may be excited by substances conveyed in the blood stream."

While these are the immediate basic physiological causes of nausea and vomiting, the specific stimuli which incite them during regional block procedures are one or a combination of the following:

Hypoxia Following Hypotension.—(see Chapters 15, 16 and 17, pages 138, 156 and 159). Since hypotension occurs most frequently following spinal and epidural blocks, nausea and/or vomiting due to hypoxia are most often seen after these regional block procedures.

The Local Anesthetic Agent.—When systemic toxic reactions to these drugs occur, nausea and vomiting are frequent (see Chapter 1 and Chapter 2, pages 6 and 33).

The Vasoconstrictor Drug.—When these drugs are used prophylactically to avoid the hypotension so often associated with spinal block and epidural block or if a systemic toxic reaction ensues from the vasoconstrictor drug which was incorporated in solutions to prolong the anesthesia, nausea and vomiting occasionally occur (see Chapter 3, page 37).

Traction During the Operation.—Traction on the viscera, particularly the esophagus, stomach, gall bladder or duodenum, results in stretching of these structures. This stretching causes stimulation of the vagus nerve, and the patient may complain of nausea and may vomit.

Premedication.—Drugs used preoperatively, particularly the opiates, may cause increased tone of the antral part of the stomach, the pyloric sphincter and the small and large intestine and thus may produce stimuli which cause nausea or vomiting.[2]

Motion and the Odor of the Operating Room.—These may effect the patient before

oxygen tank reducing valve mask and a reservoir bag (see Figure 4 page 25) or involve a complicated piece of machinery such as an anesthetic gas machine or resuscitator Artificial respiration is maintained at the rate of 16 to 20 respirations a minute The expired carbon dioxide is eliminated either by interposing a cannister containing soda lime into the system or by frequently emptying the reservoir bag Saklad[1] states In states of Tidal Hypoxia the therapeutic effort is two fold to increase the partial pressure of oxygen in the inhaled atmosphere and to increase ventilation Inhalation of high oxygen concentrations alone may not be satisfactory An increase in ventilation will not only aid in the availability of oxygen but will encourage the elimination of carbon dioxide It should be emphasized that caution must be exerted so as to eliminate only the excess CO apnea may result from eliminating it completely

Stop Convulsions—When convulsions occur from oxygen want only they are usually easily corrected by oxygen administration *Short acting intravenous barbiturates are in most instances not indicated and may actually be detrimental* They should be given only if the convulsions are not brought under control after 3 to 5 minutes of effective oxygen administration If such a situation develops then small doses of intravenous Pentothal (50 to 75 mg) may be administered at 2 to 5 minute intervals until the convulsions cease

Institute Manual Systole—If cardiac arrest or ventricular fibrillation occurs during a bout of oxygen want resuscitation of the heart as described in Chapter 7 page 74 must be instituted

Correct Posthypoxia Cerebral Edema—Often following bouts of oxygen want cerebral edema may persist and this should not be ignored Its signs and symptoms should be recognized and treatment as described in Chapter 7 page 80 instituted

Oxygen Want Following Hypertension—These cases result from prophylactic administration of vasoconstrictor drugs and are systemic toxic reactions to these drugs For treatment, see Chapter 3 page 40

Oxygen Want Following a Systemic Toxic Reaction to the Local Anesthetic Drug—See Chapter 1, page 23 and Chapter 2 page 34 for treatment of such a reaction

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quence and may be treated with oxygen, vasoconstrictor drugs, instructing the surgeon to stop visceral traction, Dramamine and/or light general anesthesia. On the other hand, if vomiting accompanies the nausea, a catastrophic situation may develop. The treatment of vomiting depends on whether or not the patient can expel the vomitus. *Regardless of which situation exists, the patient should be placed in "head-down" position (Trendelenburg) as soon as there is any evidence of nausea and/or vomiting. In this position gravity tends to prevent aspiration of the emesis.*

Treatment of the Patient Who Can Expel Vomitus.—When nausea, retching and/or vomiting occur in the patient who has the ability to expel the vomitus, the treatment of the reason for the vomiting, i.e., oxygen want, hypotension, hypertension, etc., usually suffices (for specific therapy of these complications see the chapters devoted to these problems).

Often rapid movement of the patient or the administration of intravenous drugs like ergotrate are followed by nausea and vomiting. In these cases there is no marked fall or rise in the blood pressure and the patient's condition is satisfactory except for the nausea and vomiting. Under these circumstances the administration of oxygen and/or the intravenous use of 50 mg. (1 cc.) of Dramamine will usually prove effective. The use of Dramamine in such cases has been extremely satisfactory at the Mason Clinic.

If the patient has had adequate preanesthetic treatment so that the stomach is empty, small doses (100 to 200 mg.) of Pentothal (thiobarbiturate) may be administered to anesthetize the patient lightly and eliminate the retching. However, if the patient has not been adequately prepared, this is a singularly precarious use of Pentothal, for this drug sensitizes the larynx to external stimuli and should the patient regurgitate, a severe laryngospasm may develop.

Treatment of Patient Who Cannot Expel Vomitus.—On the other hand, if the patient cannot expel the vomitus, aspiration of stomach contents into the tracheobronchial tree may take place. Rapid and correct therapy is necessary to avoid aspiration or to clear the tracheobronchial tree if aspiration occurs. Otherwise, death may result.

Opening the Mouth.—If the patient is cooperative, this may require nothing more than asking him to do so. On the other hand, an instrument may have to be used to accomplish this before the vomitus can be suctioned adequately from the mouth, pharynx, larynx and tracheobronchial tree (Figure 40, page 167).

Clearing the Mouth and Pharynx of Vomitus.—This must be accomplished by adequate suction with a large bore instrument. If the patient has not aspirated, no effort to visualize the larynx is essential. If aspiration has occurred, then the larynx must be visualized and the tracheobronchial tree cleared of vomitus. It may often be difficult to tell whether or not aspiration has occurred.[18, 19] Therefore, it has been our custom to expose the larynx and to suck out the trachea.

Visualizing the Larynx and Clearing the Tracheobronchial Tree of Vomitus.—Once the vomitus is cleared from the mouth and

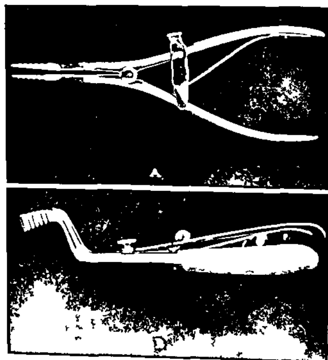


Figure 40. Roser Mouth Gag. This instrument is useful in opening a patient's mouth when the patient will not do so in the light stages of anesthesia. (A) Anterior view. (B) Lateral view.

or during the transportation to surgery or on arrival in the operating room. Motion, particularly that which results during changes in speed of an elevator, causes tension upon the esophagus and gastric walls, and these result in the feeling of nausea.[1] It should be noted that rapid, rough moving, e.g., lowering the patient from the gurney to the operating table, may simulate this elevator-like motion.

Nauseous odors, such as ether and the so-called hospital smell, may relax the abdominal wall suddenly, which will cause the lower border of the stomach to descend 1 or 2 inches and result in nausea and/or vomiting.[1]

SIGNS AND SYMPTOMS

While nausea usually precedes vomiting, either may occur separately. The patient under regional block analgesia may not complain of nausea until retching ensues. This is *singularly true in the heavily medicated patient*. Therefore, careful observation and questioning of the patient is essential throughout the block procedure if the physician is to have warning that the patient is on the verge of vomiting.

PROPHYLAXIS

Of course, the most satisfactory way to pre*vent nausea and vomiting during regional* block procedures is to avoid the stimuli which cause it and restrict the oral intake prior to surgery. But this is not always possible regardless of the care exerted by the physician. Therefore, the following precautions, which apply to any anesthetic procedure, regional or general, should be observed.

Empty Intestinal Tract Prior to Anesthesia.—The stomach at least should be emptied prior to all surgical procedures, particularly those in which spinal, epidural or intercostal splanchnic (celiac plexus) blocks are to be employed. Not to do so is asking for trouble. When the patient is being operated on for intestinal obstruction, then an attempt to empty the intestine should also be made.

Administer Drugs Known to Prevent Nausea and Vomiting.—It has been our finding as well as that of others that if Dramamine (β-dimethylaminoethyl benzohydryl ether 8-chlorotheophyllinate) is used pre- and postoperatively, the incidence of nausea and vomiting during anesthesia and the postoperative period is *markedly reduced*.[3,16] *Our dosage* orders for Dramamine are known at the Mason Clinic as the "Dramamine Routine" and consist of: 50 mg. of Dramamine intramuscularly on call to surgery, 50 mg. intramuscularly on return from surgery, and then 50 mg. intramuscularly every four hours for four doses.[4]"

Pyridoxine hydrochloride (B₆) 100 mg., Phenergan hydrochloride (promethazine hydrochloride) 12.5 mg. and d-desoxyephedrine hydrochloride (Drinalfa, Desoxyn) 5 mg. used in the same fashion as Dramamine did not approximate the reduction of vomiting seen with Dramamine. Marezine was found to be effective in reducing vomiting by Dent *et al*.[17] However, the solution of Marezine (cyclizine lactate) which was sent us and which was administered in 50 mg. doses intramuscularly was so painful on injection that after a few days trial its use had to be suspended. Chlorpromazine (Thorazine, Largactil) is not used orally or by injection pre- and postoperatively to control nausea and vomiting in our institution because of its unpredictable effect of producing severe hypotensions even in small doses (see page 141).

Have Equipment Available for Aspiration of the Mouth, Pharynx and Tracheobronchial Tree.—This includes (1) a means of suction, (2) means of opening and keeping the mouth open, i.e., teeth separator, bite block, etc., (3) laryngoscopes, (4) suction catheters and tubes, (5) bronchoscopes with sidearms for the administration of oxygen, and (6) endotracheal tubes (see Figure 3, page 23 and Figure 5, page 26). These instruments should not only be available—knowledge of their use is equally essential.

TREATMENT

Nausea without vomiting is of little conse

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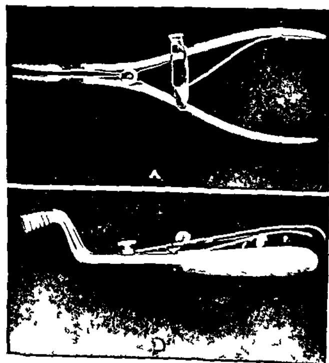


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TREATMENT

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does stress the need of having resuscitation equipment available and more important it points out that in these emergency cases the stomach and if possible, the intestine should be emptied prior to anesthesia with a Levin Ewald or a Miller Abbott tube

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pharynx the larynx can be visualized and either an endotracheal tube or bronchoscope inserted Then suction by means of a catheter or suction tube may be used in an attempt to remove the aspirated material My associates and I believe that a peek is worth two finesses and believe that bronchoscopy is the more desirable of the two methods The bronchoscope should have an arm on it for the continuous administration of oxygen during the bronchoscopy The use of the bronchoscope in contrast to the endotracheal tube technique allows direct visualization of the tracheobronchial tree and *assures* removal of large food particles which might obstruct the trachea or bronchi

Giving Oxygen —As soon as the vomitus is aspirated give oxygen by bag and mask If the patient has stopped breathing use a positive pressure method of giving oxygen via a closed system Administering oxygen prior to the removal of vomitus only pushes the material more deeply into the tracheobronchial tree It should be remembered that once the airway is cleared oxygen may be given through the sidearm of the bronchoscope or the bronchoscope may be removed and an oral airway or an endotracheal tube inserted in its place If the bronchoscope is removed a cuffed endotracheal tube is preferable to an oral airway because once the cuff is inflated one avoids further contamination of the tracheobronchial tree should vomiting recur The cuffed endotracheal tube also prevents pumping oxygen into the esophagus and stomach Acute gastric dilatation alone has been known to cause death

Administering Antibiotics —Following aspiration of stomach contents the membrane lining the alveoli is usually damaged by the hydrochloric acid in the gastric juices For this reason antibiotic therapy should be instituted immediately to combat secondary bacterial invasion of the lung parenchyma

Associated Therapy —If hypotension cardiac failure etc occur concomitantly with this complication they must be treated as indicated in the chapters of this book dealing specifically with these problems

COMMENT

General vs Spinal Anesthesia for Intestinal Obstruction —It should be noted that spinal anesthesia has been advocated as the anesthesia of choice in intestinal obstruction probably because the patient retains his laryngeal and tracheal reflexes and therefore theoretically should be able to prevent aspiration if he regurgitates Graham and Brown[40] even go so far as to state "A surgeon who operates upon a patient suffering with acute intestinal obstruction using inhalation anesthesia if adequate facilities for spinal anesthesia are available is guilty of malpractice" This may or may not be true and it is not the purpose of this section to argue whether spinal or general anesthesia is the procedure of choice in an intestinal obstruction *However when a patient who has a high spinal anesthesia (a level of T_4 or higher) and who is acutely ill (most intestinal obstruction cases are) vomits HE MAY NOT BE ABLE TO EXPEL THE VOMITUS FROM THE PHARYNX* In these cases even though the vocal cords (which form the "watchdog of the larynx) close the abdominal musculature against which the diaphragm pushes to effect a forceful expiration and cough is paralyzed by the spinal and the pharyngeal musculature and tongue may not be able to expel the amount of vomitus rapidly merely by a spitting action Therefore as the patient becomes more hypoxic he eventually takes a deep inhalation and although awake may drown in his own vomitus if it is not aspirated by suction When a conscious patient finds himself in this situation he may become completely uncooperative and as he becomes more irrational from the hypoxia and as the CO_2 accumulates he may resist any attempt either verbal or mechanical to open his mouth so that the suction can be used This makes the job of re establishing a patent airway very difficult and if the vomitus cannot be removed the patient may die

The foregoing does not preclude the use of regional block in intestinal obstruction The author often employs it Nevertheless it

(2) decrease in the subarachnoid space (intra-abdominal pressure causes pressure on the vena cava which in turn results in distention of the veins in the epidural space, and this at least theoretically, decreases the amount of spinal fluid the lumbar part of the subarachnoid space may contain), or (3) unknown factors

Relative Overdosage.—Occasionally the dosage the number of milligrams and dilution as well as the site of the lumbar tap may be accurate but other factors, some known and others unknown may result in a high and/or total spinal

Among the *known factors* are these (1) positioning of the patient (2) failure of the local anesthetic agent to "fix" in 20 minutes (3) speed of injection (4) barbotaging * and (5) the increase in spinal fluid pressures and presumably in its circulation caused by straining coughing deep breathing and labor pains For example if a steep Trendelenburg position is used immediately following the injection of a hyperbaric solution (a solution *heavier than spinal fluid*) *the anesthetic level* may rise to a high level rapidly while with the same dosage without tilt of the table it would in all probability not rise above the desired level If the solution is injected rapidly or barbotaged—two methods of increasing the circulation of the spinal fluid—the level may also rise to an unexpected height

It has also been mentioned that in cases where the spinal fluid is diminished because of severe dehydration a normal or small dosage of the local anesthetic solution may result in a high or total spinal Kamsler *et al*[6] report several incidences of massive spinal anesthesia in severely dehydrated patients following 15 mg of Metycaine given by the continuous technique of Tuohy Since dehydration was the only common factor in all these cases they concluded that (1) the agent injected was not sufficiently diluted by the diminished spinal fluid (2) neural tissue is more susceptible when dehydrated or (3) other indeterminate factors associated with dehydration may have produced the results However, it is doubtful that diminished spinal fluid will result in massive spinal anesthesia for at the Mason Clinic, the day prior to surgery a myelogram is often performed to confirm the diagnosis of ruptured nucleus pulposus At this time a reasonably large amount of spinal fluid is removed when an attempt to recover the Pantopaque is made

* Barbotage is the injection of a portion of the prepared local anesthetic solution into the subarachnoid space then the withdrawal of more spinal fluid into the syringe injection of a portion of this resultant mixture withdrawal of spinal fluid again injection and so on decreasing the amount of spinal fluid withdrawn each time until finally all of the contents of the syringe have been injected [5]

TABLE XI

REPORTED INCIDENCE OF HIGH OR TOTAL SPINAL ANESTHESIA FOLLOWING EPIDURAL (PERIDURAL) BLOCK

Site of Injection	*Author*	*Percentage*
Caudal region	Moore*	2 in 3500 blocks = 0 06% (In 6 cases spinal fluid was obtained and the needle was readjusted before injection)
	Bonica[2]	6 in 4200 blocks = 0 14% (Spinal fluid was noted in an additional 0 7% of cases and the needle was readjusted before injection)
Lumbar or thoracic region	Moore*	0 in 1700 blocks (The dura was punctured in 15 cases the needle was moved to a different interspace and an epidural block was administered)
	Bonica[2]	1 in 2290 blocks = 0 04% (The dura was punctured in 75 cases In 15 cases intentional spinal blocks were executed in remaining 60 cases the needle was moved to a different interspace and an epidural block was administered)
	Selva de Assis[3]	2 in 1000 blocks = 0 2%
	Bromage[4]	1 in 1000 blocks (approx) = 0 1%

* Author's series at the Mason Clinic

CHAPTER 19

High and/or Total Spinal (Subarachnoid) Block Following Spinal and Epidural Block

A HIGH or total spinal will often follow a *purposely* administered spinal or epidural block and therefore should not surprise the physician or result in a catastrophe The term high spinal is often used synonymously with the term "total spinal although by definition they are different A "high spinal occurs when the level of a spinal anesthetic rises to the upper thoracic or cervical dermatomes On the other hand a total spinal occurs *when the local anesthetic agent rises above* the level of the foramen magnum

ETIOLOGY

High or Total Spinal Following an Epidural Block—The most probable reason that a high or total spinal anesthesia may develop after an epidural block is that the dura is inadvertently punctured and the local anesthetic drug is injected into the subarachnoid space However Frumin *et al*[1] found that following epidural anesthesia with 2% Novocain in man the concentration of Novocain in the spinal fluid during the anesthesia was about 0 2 mg per cc This they state is the threshold concentration of this drug for the production of spinal block They concluded that at least part of the anesthesia during the epidural block technique in man is the result of dural penetration of the Novocain and the subsequent production of spinal anesthesia According to this work as well as other experiments cited by *Bromage*[2] which seem to substantiate the conclusions of Frumin *et al*[1] it is theoretically possible that a high spinal block may follow an epidural block without puncture of the dura It is the opinion of the author and his associates from observing approximately 1700 such blocks at the Mason Clinic that even though this possibility exists in reality a high spinal anesthesia seldom if ever follows an epidural block unless the dura is tapped

The reported incidence of high or total spinal following an epidural block is small (see Table XV page 171)

High or Total Spinal Following Spinal Block—When this complication follows a purposely administered spinal anesthesia the cause is overdosage However the cause may not always be an *actual* overdosage of the drug but a *relative* overdosage i e, the dosage of the local anesthetic solution may be well within normal limits but other factors cause it to rise to an unpredictable height

Actual (True) Overdosage—In this instance either the amount of the drug i e number of milligrams or the volume to which it is diluted may be excessive or the volume and the milligrams may be accurate but the site of the spinal tap too high say at T_{12} or L_1 instead or L_3 or L_4

It should be emphasized that whenever the intra abdominal pressure of a patient is increased for example by obesity pregnancy abdominal tumors or ascites the dosage of the local anesthetic drug and its solvent must be reduced Our experiences at the Mason Clinic prove the validity of this consideration although it is difficult to determine whether this effect is due to (1) the flattening of the lumbar curvature (see Figure 45 page 198),

means then of knowing whether the motor roots of the phrenic are affected, or whether some of the anesthetic solution has entered the fourth ventricle and reached the respiratory centre located there "

Circulatory Changes —Initially the blood pressure and pulse may remain normal As more of the sympathetic nervous system is blocked the blood pressure drops from *dilatation of the vessels* and *oxygen want* develops first from hypotension and then from paralysis of the muscles of respiration The pulse becomes more rapid and feeble If the problem is not treated at this point the blood pressure and the pulse become unobtainable

Appearance of the Patient —The skin may show little change or the patient may look cadaveric The mucous membranes particularly the lips are blanched and pale Cyanosis seldom occurs even if there is 5 gm of unreduced hemoglobin per 100 cc present in the circulating blood because peripheral vasoconstriction in the unanesthetized area (an attempt by the body to maintain adequate circulation) prevents the blood from reaching the subcutaneous tissue and skin of the upper extremities neck and head

Death —If the above signs and symptoms of a high or total spinal anesthesia are not immediately recognized and treated cardiac failure and death may ensue

PROPHYLAXIS

Steps to prevent a high or total spinal after spinal and epidural block depend mostly on which block is being performed However the one precaution which is applicable to both is *the careful and constant observation of the patient* after the block If this precaution is not taken and the block is performed in a nonchalant manner high spinals will go unnoticed more easily and result in death The physician must at all times check the dermatome level anesthetized the blood pressure and the pulse Of course the surgeon cannot properly attend to these himself Graham and Brown[9] state, "We cannot too strongly condemn the practice of a surgeon being his own anesthetist The technique of administering the anesthetic is important but of no more importance than the preparation of the patient for the anesthetic or the intelligent supervision of the patient throughout the whole operative procedure Spinal anesthesia is a dangerous anesthetic agent unless administered by an expert anesthetist trained in spinal anesthesia"

Spinal Block —*When Doing a Spinal Block in an Obese or Obstetrical Patient, Reduce the Dosage of the Drug* —In the obese patient the pregnant and other instances where the intra abdominal pressure is increased the dosage i e the amount of the local anesthetic drug and the hypertonic dextrose solution if used, as well as the total volume to which these drugs are diluted with spinal fluid should be reduced by ⅓ to ½ of the usual dose[10] In these patients even with these reduced dosages the level of anesthesia rises rapidly and to heights usually unobtainable in the normal adult with such small amounts

Correct Low Spinal Fluid Pressures —If a spinal tap for diagnosis has been performed (myelography etc) prior to surgery or if the patient is markedly dehydrated the patient should be placed on a hydration routine (see Chapter 20 page 184) before a spinal anesthetic is given if time permits This may facilitate the spinal tap at the time of surgery and may prevent a high spinal anesthesia of the type described by Kamsler *et al*[6]

Correct Deficiencies in Blood Volume —If possible prior to administration of a spinal anesthetic every effort should be made to see that any deficiencies in blood volume are corrected A low blood volume may render it extremely difficult to treat the hypotension and oxygen want associated with a high spinal anesthesia (see Chapter 15, page 139)[11 12]

Do Not Inject New Local Anesthetic Drugs into the Subarachnoid Space Unless Their Dosages and Physiological Characteristics Have Been Definitely Established by Careful Research —Recently Xylocaine (lidocaine) and Cyclaine (hexylcaine) have been introduced for spinal block usage The author has not used either drug intrathecally but Hoyt[13]

This plus the normal leakage of spinal fluid over the next 12 to 24 hours through the hole made in the dura by the 18 gauge needle often results in the presence of only a minimal amount of spinal fluid at the time spinal anesthesia is given for the surgery In these cases we have had no trouble with resulting high or total spinal anesthesia although a dose of 10 mg of Pontocaine and 1 0 cc of 10% dextrose diluted to a volume of 3 cc with spinal fluid is injected at L_ On the contrary at times we have had difficulty establishing adequate anesthesia in these cases, evidently because of the hypotension of the spinal fluid and some of these patients have had to be reblocked to get a level of analgesia above T_{11}

In the second category i e, *the unknown factors* are those physiological properties of a patient s spinal fluid which can not be determined prior to the administration of every spinal anesthetic and include (1) the rate of diffusion of the local anesthetic agent in the cerebrospinal fluid (2) the rate of the circulation or the convection of the cerebrospinal fluid and (3) its alkalinity i e the pH of the cerebrospinal fluid

SIGNS AND SYMPTOMS

Whether the high or total spinal anesthesia is a result of a purposely administered spinal anesthesia a misplaced needle during an epidural block or the complication of a peripheral nerve block the signs and symptoms are as follows

Mental Manifestations —The patient becomes drowsy does not respond rapidly to questions and may appear to be comatose Macintosh[7] states Purdon Martin has pointed out that consciousness is maintained by an awareness of the body and of the environment Consciousness lapses in the absence of sensory impulses to the cortex It is clear and the writer [Macintosh] can testify from personal experience that the higher a spinal anesthetic spreads the more the awareness of the body is diminished The area from which the cortex receives stimuli is lessened If the solution spreads to the foramen magnum sensory stimuli from the body are eliminated and the patient remains awake by virtue of the stimuli reaching the cortex from the cranial sensory nerves"

This explains why drowsiness occurs from high spinal anesthesia and why the anesthetic drug must spread into the cerebrospinal fluid above the foramen magnum paralyzing the cranial nerves and brain before the patient becomes unconscious

Pupils —The pupils retain their normal size constrict or dilate according to the type of premedication If the high or total spinal anesthesia goes untreated they dilate as death approaches

Oxygen Want —As the spinal anesthetic rises above the level of the fourth thoracic vertebra the patient may yawn and complain of dyspnea but if he is heavily medicated or if the rise is rapid these symptoms are absent The usual signs of impending respiratory paralysis are (1) diminution of the intercostal muscle activity with increasing diaphragmatic excursion (2) loss of the spoken voice (3) dilatation of the alae nasae and (4) the use of the accessory muscles of respiration of the neck with dropping of the jaw with each respiration[8] When the anesthetic solution rises above the foramen magnum diaphragmatic action usually ceases and the patient becomes unconscious In the rare case twitching of the muscles around the mouth and eyes may be noted indicating the onset of convulsive movements from hypoxia or anoxia Macintosh[7] writes It is well known that in local and spinal analgesia sensory nerve fibers are affected before motor If the concentration of the anaesthetic is low enough there can be sensory loss without motor paralysis In really high spinal analgesia the skin over the neck (C2 3 4) can be insensitive when the diaphragm supplied by the motor roots of the phrenic (C3 4, 5), still functions When the spinal analgesia is more extensive still the patient loses consciousness Even now diaphragmatic activity may be enough to sustain life On the other hand respiration may cease and there is no

On the other hand high or total spinal block following what was intended to be a low spinal epidural or peripheral nerve block is not expected and thus brain damage or death may result[16] However if the patient has been carefully observed following the block the complication should be recognized early and there should be no cause for alarm excitement or chaos In most cases neither a high nor total spinal anesthesia should be followed by morbidity or mortality if it is recognized immediately and the physician maintains his composure so that he may institute treatment effectively

Irrespective of whether a high or a total spinal anesthesia results the treatment is essentially the same and must be instituted immediately and correctly

Correct the Hypotension —Intravenous fluids should be started and vasoconstrictor drugs given intravenously to prevent or correct hypotension (see Chapter 15 page 147 for specific drug therapy)

Prevent Oxygen Want—Although oxygen want in these cases is initially a result of the hypotension which results from dilatation of the blood vessels in the anesthetized area the treatment of oxygen want must be started simultaneously with efforts to raise the blood pressure because the nerves to the muscles of respiration are also paralyzed If the patient stops breathing or his effort to aerate his lungs *is ineffectual artificial respiration* must be instituted immediately The use of a bag and mask through which oxygen can be given under pressure is the most efficient and effective means of combating oxygen want If this equipment is not immediately available manual artificial respiration or mouth to mouth breathing should be instituted *Barbiturates and analeptics are contraindicated! If convulsions occur they are due to hypoxia or anoxia and will be corrected by oxygenation of the patient*

If *nausea* and/or *vomiting* result from hypoxia the airway must be cleared by tracheal suction and/or bronchoscopy prior to administering the oxygen (see Chapter 17 page 167) The most effective way to maintain a patent airway is to insert an endotracheal tube

Artificial respiration must be maintained until the anesthesia recedes and the patient can maintain his own respirations

Withdraw Spinal Fluid —If an inadvertent spinal administration following an epidural block is recognized early and the needle is in the subarachnoid space at the time as much spinal fluid as possible should be removed immediately remembering that too rapid withdrawal may result in herniation of the brain stem through the foramen magnum Withdrawal may prevent a high or total spinal altogether or at least cause it to terminate more rapidly

However if the needle has been removed (as is usually the case during a purposely administered spinal anesthesia) and a high or total spinal occurs no attempt to remove spinal fluid should be made until the *oxygen want* and *hypotension* are under control To do so is a waste of valuable time and may result in the death of the patient The value of withdrawing spinal fluid in this situation i e after 15 to 30 minutes has elapsed is probably negligible

Treat Cardiac Failure —If cardiac failure occurs it must be treated as indicated in Chapter 7 page 74

COMMENT

High Epidural Block —Following a purposely administered epidural block the anesthesia may occasionally reach a high unexpected level and the resulting respiratory depression hypotension and drowsiness resemble the signs and symptoms of a high spinal anesthesia This is clearly one of the *risks* of epidural block Nevertheless since the dura fuses with the inner layer of the periosteum of the skull at the foramen magnum the vital centers in the brain are not bathed by the anesthetic solution and the patient does not become unconscious Treatment in these cases is essentially the same as for high spinal and consists of preventing oxygen want raising and maintaining blood pressure and waiting for the high level of anesthesia to recede

reports small doses of Cyclaine heavily weighted with dextrose which have been used to produce either saddle or low spinal analgesia in obstetrics have a tendency to rise for an unpredictable period of time Hoyt found in his obstetrical patients that after 20 minutes he might have skin anesthesia to the tenth or twelfth thoracic dermatomes only to find at the end of the delivery that the sensory level had risen to the fourth thoracic dermatome or higher This makes it clear that the new local anesthetic drugs for spinal analgesia should be tested only by physicians who are adept in the art of resuscitation One must not forget Pfeiffer's[14] declaration We decline to subject patients to each new venture in the pharmaceutical line"

Adherence to the Technique—At all times the physician must closely adhere to the steps of the specific technique selected He should be particularly cognizant of whether the solution he is using is hypobaric (lighter than the specific gravity of spinal fluid) isobaric (same specific gravity as the spinal fluid) or hyperbaric (heavier than spinal fluid)

Miscellaneous—Most authors advise against the injection of a local anesthetic solution into the subarachnoid space during a labor pain or other acts of straining which are thought to cause the anesthesia to rise to abnormal heights[15]

Likewise barbotage may not be advisable because it too can cause the anesthesia to rise to an unwanted height

Epidural Block—*Observe the Needle for Drops of Spinal Fluid Appearing at Its Hub*—Once the needle is in position for an epidural block and a 2 to 3 cc test dose has been injected the hub should be carefully watched for 15 to 30 seconds for the appearance of spinal fluid before the remaining contemplated dose of the drug is injected During an epidural block it is not unusual after the test dose for fluid to drip from the needle If this does not cease after the first few drops then the fluid should be allowed to drop on the inside of the physician's forearm If it is cold it is probably the anesthetic solution and if it is warm it is spinal fluid[17]

Chemical tests may also be of value when fluid drips from the needle Bromage[4] states The drops can be collected in a test tube containing a reagent which will give a specific colour change when the analgesic solution used is added to it A colour change in the reagent then indicates that the liquid dripping back is undiluted epidural solution (local anesthetic solution) and not spinal fluid

For example procaine can be detected by means of the diazo reaction because it contains an aromatic amine group A solution of nitrous acid (produced by mixing sodium nitrite with hydrochloric acid) and B naphthol changes from yellow to red on the addition of procaine" However he points out that testing the drops on the forearm is quite adequate and does not require added assistance"

Aspirate Frequently—While injecting the local anesthetic solutions during epidural blocking frequent attempts to aspirate at least in two planes and preferably in four planes should be made to be sure the needle has not been advanced into the subarachnoid space

Inject a Test Dose—When performing an epidural block it is always wise to inject a test dose of 2 to 3 cc of the solution of the drug wait for 5 minutes and then test for unusual or extensive areas of analgesia before injecting the balance of the contemplated dose[17] If puncture of the dura is signalled by an extensive area of analgesia but one inadequate for the surgical procedure a small additional amount of the solution may be given in an attempt to obtain the desired height or to be completely safe the block should be discontinued and the existing analgesia supplemented with a general anesthetic

TREATMENT

If the physician administering the anesthetic intends to produce a high spinal anesthesia or to induce *hypotensive anesthesia* and he is carefully watching the patient for sequelae like hypotension and oxygen want their onset will not cause alarm and morbidity or mortality will seldom result

Headache

Persistent headaches following regional block analgesia are not always precipitated by the block technique alone, i.e., the actual placement of the needle or needles and/or the injection of the local anesthetic solution. Headaches may be arbitrarily classified for the purposes of this book into two groups, i.e., those unassociated and those associated with puncture of the dura.

Headaches Not Associated with Puncture of the Dura.—This type of headache has the following characteristics: (1) it is felt irrespective of the position of the body; (2) it is not necessarily localized but occurs in the frontal region, the occipital region, or the top of the head; (3) its onset varies—it may come at the time of administration of the anesthetic, during the operation, or in the period of convalescence; (4) it is of mild or moderate intensity, usually easily controlled by mild analgesic drugs such as acetylsalicylic acid (Aspirin); and (5) it is subject to remissions between episodes irrespective of body position.

This type of headache is precipitated by mental irritation, mental and physical fatigue, position during operation, climacteric, fever, menstruation, anxiety, etc., and usually occurs in patients who are predisposed to headaches (migraine, etc.). It is *not* related to the anesthetic procedure.

Example I. Occasionally following cervical block for a thyroidectomy the patient may complain of an occipital headache which may persist for one to two days. In such cases the headache should not be blamed on the block procedure: the identical type of headache may follow the use of general anesthesia for this surgical procedure. The headache and neck pain are due to hyperextension of the neck muscles due to a position used by many surgeons for thyroidectomy.

Example II. When a bilateral oophorectomy with or without hysterectomy is performed, particularly in a young female, the menopausal symptoms from castration often include an intermittent throbbing headache which may last indefinitely, i.e., 1 to 2 years. Often when a headache of this type occurs it is automatically attributed to the anesthetic procedure, particularly if a spinal anesthesia has been administered. This assumption is unwarranted.

Frequently headache may occur following general anesthesia, no anesthesia at all, or at the climacteric.[1,5] Maxson[5] states: "While headache still remains the most frequent aftereffect [of spinal anesthesia], modern technique has reduced its incidence so that its occurrence is less frequent than after the administration of ether." Other studies substantiate the belief that the incidence of headache is no greater after spinal anesthesia than after inhalation anesthesia but that following spinal block it may be more troublesome.[1,2] My associates and I have more than once been notified by a surgeon that a patient had a "spinal headache" only to discover from the chart that the patient had not had this type of analgesia, but that on the contrary, intravenous Pentothal (thiopental) or an inhalation anesthetic had been administered. Arner[4] cites similar experiences.

Other causes of headache following regional block analgesia which are not caused by dural punctures are those associated with administration of vasoconstrictor drugs. These are noted elsewhere (see Chapter 3, page 38).

Headache Associated with Puncture of the

REMEMBER THAT THE TREATMENT OF A HIGH OR TOTAL SPINAL ANESTHESIA IS ESSENTIALLY THE TREATMENT OF HYPOTENSION AND OXYGEN WANT, AND THE CHAPTERS DEVOTED TO THESE, I E, CHAPTER 15, PAGE 138 AND CHAPTER 17, PAGE 159, SHOULD BE REVIEWED

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Number of Holes in the Dura—The greater the number of holes made in the dura during an attempt at lumbar puncture the more likely is a rapid loss of spinal fluid with consequent headache

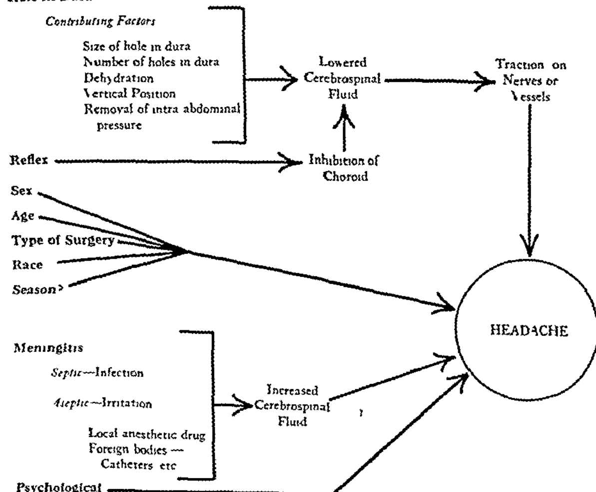
Dehydration—Dehydration prior to or following puncture of the dura results in a lack of body water for the choroid plexus to draw on in forming new cerebrospinal fluid Therefore the rate of formation of spinal fluid may not equal the leakage via the hole in the dura

Vertical Position—Early ambulation or elevated positions in bed to facilitate the recovery of a surgical or obstetrical patient before the hole in the dura has closed would automatically augment the leakage of spinal fluid reduce the volume of the cerebrospinal fluid and result in a subnormal cerebrospinal fluid pressure with the possibility of headache

Removal of Intra abdominal Pressure—When intra abdominal pressure is relieved by the removal of a tumor the delivery of a baby or the aspiration of ascitic fluid there may be a decrease in the size of the epidural (peridural) and meningeal veins in the lumbar area which in turn results in an increase in the epidural space This may allow the dural sac to accommodate more cerebrospinal fluid lowering the intracranial cerebrospinal fluid pressure and lessening its cushioning effect on the brain This mechanism would be the opposite of that which operates when

TABLE XVI

CAUSES OF SPINAL HEADACHES
(*Adapted from Owen et al*[30])



Dura—There are two distinct types of headache which may follow spinal taps one is due to an increase in spinal fluid pressure and the other is caused by a decrease

Headache from Increased Spinal Fluid Pressure—The headache in these cases is a result of the increased formation of spinal fluid or a decreased absorption of it by the leptomeninges It is not relieved or potentiated by changes in position This type of headache is most often associated with meningeal irritation and will be discussed under MENINGITIS Chapter 22 page 202

Headache from Decreased Spinal Fluid Pressure—This is the type most frequently encountered following puncture of the dura and it may be termed 'hypotensive spinal headache *It is the one with which the rest of the material included in this chapter deals*

ETIOLOGY OF HEADACHE FROM HYPOTENSION OF THE SPINAL FLUID

Headache is probably the most common complication of dural tap whether it be performed for spinal (subarachnoid) block or diagnostically[1,40] According to articles on spinal anesthesia it occurs in 1 to 30% of the patients receiving this type of anesthesia The relatively wide variation in its incidence evidently depends on the technique of individual authors and their criteria as to which headache is and which is not caused by the lumbar puncture Its occurrence has been known since the time Bier[6] and Corning[7] first used this technique

At the present time it is agreed that most spinal headaches are caused by leakage of spinal fluid through the hole in the dura at a rate that exceeds its replacement by the choroid plexus However sex age type of surgery local anesthetic agent race weather inflammation dural reflex with spasm of the choroid and mental disturbances have been mentioned as possible contributing factors (see Table XVI page 179)

Leakage of Spinal Fluid—The fact that a low spinal fluid pressure exists in most but not all patients with postlumbar puncture headache has been definitely established[8,41,45] However the corollary to this statement that patients with a low spinal fluid pressure following lumbar tap have headaches does not follow[44] The degree to which the spinal fluid pressure must be lowered to create a headache is not definitely known However Franksson and Gordh,[34] after attempting to measure the amount of fluid escaping through a hole of average size in the dura estimated that as much as 240 cc may be lost in a 24 hour period

The length of time a headache from leakage of spinal fluid persists depends to some extent on the length of time it takes the dura to heal and it has been shown that a hole in the dura may not heal for six days to two weeks[2-,34,46] Pool[46] following myeloscopic examinations of living patients states The myeloscopy has frequently revealed large collections of epidural fluid when ordinary lumbar puncture has been done within the previous two to four days This demonstrates that leakage of spinal fluid can continue for some time after the ordinary lumbar tap My associates and I can confirm this statement by Pool from our experience in performing 75% of the myelograms at the Mason Clinic It is of some interest to note that Schube and LeDrew[47] believe that spinal headaches occur and persist only if the arachnoid membrane herniates or is pulled through the hole in the dura The herniation of the arachnoid membrane might explain why the hole in the dura may not close

Factors which augment hypotension of spinal fluid are the size of the holes in the dura the number of holes dehydration vertical position and removal of the intra abdominal pressure

Size of Hole in Dura—It is obvious that the larger the hole in the dura is the more rapidly spinal fluid escapes and the longer it takes for the dura to repair itself The lowering of the incidence of spinal headache by the use of the 22 or smaller gauge needles is conclusive *evidence of this*[4,10,15,20,31,34,48,51,104,105]

Number of Holes in the Dura —The greater the number of holes made in the dura during an attempt at lumbar puncture the more likely is a rapid loss of spinal fluid with consequent headache

Dehydration —Dehydration prior to or following puncture of the dura results in a lack of body water for the choroid plexus to draw on in forming new cerebrospinal fluid Therefore, the rate of formation of spinal fluid may not equal the leakage via the hole in the dura

Vertical Position —Early ambulation or elevated positions in bed to facilitate the recovery of a surgical or obstetrical patient before the hole in the dura has closed would automatically augment the leakage of spinal fluid reduce the volume of the cerebrospinal fluid and result in a subnormal cerebrospinal fluid pressure with the possibility of headache

Removal of Intra abdominal Pressure —When intra abdominal pressure is relieved by the removal of a tumor, the delivery of a baby or the aspiration of ascitic fluid, there may be a decrease in the size of the epidural (peridural) and meningeal veins in the lumbar area which in turn results in an increase in the epidural space This may allow the dural sac to accommodate more cerebrospinal fluid lowering the intracranial cerebrospinal fluid pressure and lessening its cushioning effect on the brain This mechanism would be the opposite of that which operates when

TABLE XVI

CAUSES OF SPINAL HEADACHES

(*Adapted from Owen et al*[30])

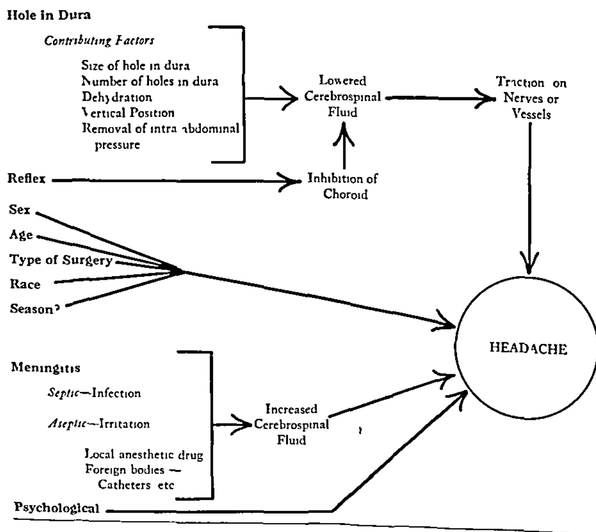


TABLE XVII

INCIDENCE OF POSTLUMBAR PUNCTURE HEADACHE FOLLOWING INITIAL SPINAL ANESTHESIA IN 997 PATIENTS

Analysis with Respect to Sex, Age and Different Puncture Needles

(From Arner[4])

Sex	*Age in Years*	*Postlumbar Puncture Headache* — *Antoni's Needle [double needle]*	*Pitkin's Needle*	*Both Needles*
Males	Under 31	3.3% (5/152)	12.5% (6/48)	5.5% (11/200)
	31–50	4.7% (8/172)	5.8% (7/121)	5.1% (15/293)
	Over 51	0.5% (1/208)	1.0% (1/105)	0.6% (2/313)
	All ages	2.6% (14/532)	5.1% (14/274)	3.5% (28/806)
Females	Under 31	11.8% (2/17)	0.0% (0/12)	6.9% (2/29)
	31–50	6.3% (2/32)	8.3% (3/36)	7.4% (5/68)
	Over 51	4.1% (2/49)	6.7% (3/45)	5.3% (5/94)
	All ages	6.1% (6/98)	6.5% (6/93)	6.3% (12/191)
Males and Females	Under 31	4.1% (7/169)	10.0% (6/60)	5.7% (13/229)
	31–50	4.9% (10/204)	6.4% (10/157)	5.5% (20/361)
	Over 51	1.2% (3/257)	2.7% (4/150)	1.7% (7/407)
	All ages	3.2% (20/630)	5.4% (20/367)	4.0% (40/997)

The figures in parentheses denote the number with headache in relation to the number observed

extra abdominal pressure is used to relieve headache (see page 191)

Sex.—Headaches seem to be more common in the female than in the male (see Table XVII page 180)

Age.—In the young vigorous patient between the ages of 10 and 50 the incidence of headache seems to be higher (see Table XVII page 180, and Table XVIII page 181)[4, 20, 28, 34]

The reason for the decrease of headache in patients above fifty is somewhat obscure but if the current theory is correct, namely that headache is caused by downward movement of the brain and stretching of its meninges and blood vessels then it should be expected to occur less in the older patient since the blood vessels are less susceptible to stretching with increasing age Dripps and Vandam[83] state "There is a numerical reduction in the number of pain fibers to an area with age Age also brings with it a reduction in elasticity of blood vessels" Owen *et al*[30] suggest that "Another enigma [of spinal headache] is the greater frequency in young adults and females The meninges of such individuals may be more elastic and thus more susceptible to dilatation and traction"

Type of Surgery.—The general clinical impression has been recorded that following vaginal obstetrical deliveries anorectal and pelvic surgery the incidence of spinal headache is higher than following other surgical procedures[15, 16, 17, 30, 51, 5] Whether it is actually the type of surgery the age of the patient (most obstetrical patients are young) the sex of the patient and the pre and postoperative management i e early ambulation dehydration etc or a combination of these would be most difficult to determine

Greene[15] writes Today the need for minimal dural punctures is greater than ever before Early ambulation following operation and parturition increases the incidence of headache after spinal anesthesia The assumption of the erect position before the dural opening has closed augments the leakage of spinal fluid and allows the reduced volume of cerebrospinal fluid to manifest itself as subnormal intracranial pressure with headache as the result In obstetrics the complication is even more common and severe Intra abdominal pressure is greatly reduced after delivery and therefore the patient lacks the

TABLE XVIII

HEADACHES PER 100 PATIENTS OF EACH AGE GROUP
(*From Krueger*[20])

Age group	Headaches per 100 patients
Under 20 years (243 patients)	11.5%
20-35 years (6[illegible]0 patients)	12.6%
36-50 years (97 patients)	9.2%
Over 50 years (127 patients)	2.4%

usual support to intraspinal fluid pressure provided by a normally tonic abdominal wall. Blood loss and dehydration are more marked and less treated among postpartum patients than among routine surgical cases. The effect of water balance is to decrease the availability of water for the production of cerebrospinal fluid at a rate sufficient to reproduce the estimated daily loss of 240 cc of spinal fluid through a dural puncture of average size. The puerperal convalescent is especially prone to complain of postspinal headache not only because, as a woman, she is more likely to feel and express the complaint but also because she usually does not expect to have any serious discomfort after the first or second day of the puerperium. For these reasons the incidence of headache following spinal analgesia for vaginal delivery has been reported to be greater than in any other group of hospital patients ranging from 15 to 30% depending on the diameter of the spinal needle, the earliness of postpartum ambulation and the type of patient.

Krueger[20] states: "There was a much higher incidence of headache in obstetric patients than in those having general surgical procedures.

"The obstetric patients, being younger on an average than the general surgical patients, made it look as though there was a direct relationship between age and resistance to *postspinal headache*.

"The difference in the incidence of headaches in the two groups may be explained by the following factors:

(1) The general surgical group includes many poor risk patients who are bedfast for several days postoperatively, thus decreasing early leakage of spinal fluid.

(2) The use of narcotics and analgesics in patients having general surgical procedures might be expected to dull any headache that might be present.

(3) The operative pain, general malaise and lack of cerebral acuity from arteriosclerosis all help to prevent a postspinal headache from being noticed or reported. These factors are not so prevalent in obstetric patients as in patients who have general surgical procedures.

(4) Most of the general surgical patients received 5% dextrose in distilled water intravenously in the operating room (such is not the case with our obstetric deliveries) and fluid balance was checked on the wards, so that dehydration did not occur."

Owen et al[30] claim that "The high incidence of headache after obstetric, anorectal and pelvic surgery may be related to postoperative dilatation of the pelvic veins with compensatory collapse of the epidural veins and dilatation of the dural sac. This would reduce the amount of spinal fluid available for floating the brain and thus intensify spinal headache."

Race.—Race has been mentioned as a possible factor for headaches following puncture of the dura.[28, 5-] Grady *et al*[35] found a difference in distribution of headaches according to race (see Table XIX, page 182).

TABLE XIX

HEADACHES—RACE AND SEX DISTRIBUTION OF PATIENTS
(*After Grady et al*[35])

	No of Cases	*No of Head aches*	*Per cent in Each Group*	*Per cent of 28 Headaches*
White females	625	17	2 72	60 7
White males	781	8	1 02	28 9
Colored females	488	0	0	0
Colored males	406	3	0 74	10 7

Dural Reflex —This has been mentioned as a possible cause[54] It would as Owen *et al*[30] point out seem nebulous On the other hand if irritation can produce vascular spasm in an extremity, which results in pain perhaps it may produce it in the vessels of the brain with consequent headache

Psychic Disturbance, Lack of Physical Stamina, etc —These have been suggested as specific causes of hypotensive spinal headache[29, 57, illegible] The fact that the psychological make up of a patient may perhaps influence the severity of interpretation of a headache is not doubted and it may be a "potentiating factor but its importance as a specific cause is doubtful The response to posture abdominal compression hydration and subarachnoid and epidural saline etc indicates that a spinal headache is in most instances not imaginary

SIGNS AND SYMPTOMS OF HEADACHE FROM HYPOTENSION OF THE SPINAL FLUID

Postural headache is the most common of the signs and symptoms of leakage of spinal fluid from a puncture hole in the dura However the patient may also complain of nuchal rigidity or pain i e a stiff neck visual disturbances tinnitus and decrease in hearing

Postural Headache —*Onset and Duration* —The headache appears 1 to 3 days after the lumbar puncture and usually persists for 1 to 5 days In a few instances however the headache may not develop for 7 to 10 days and may last for weeks or months[33] The author has seen this type of headache last up to 30 days

Characteristics —The outstanding characteristic of this type of headache is that it is brought on and aggravated by sitting or standing and is relieved by lying flat In most instances *it is not noticed until the patient has* assumed the vertical position for a period varying from two hours to two days[0] It is usually described as being occipital bifrontal or orbital but may be located any place in the calvarium (see Table XX page 184) When the patient gets out of bed the headache is usually minimal and tends to increase in severity as the patient remains erect Rapid shaking of the head or jugular compression will aggravate the headache while compression of the common carotids will partially alleviate it

Mechanism —Wolff[56, 57] points out that changes from the normal in the following intracranial structures may result in painful impulses (1) the venous sinuses and their tributaries (2) part of the dura mater at the base of the brain (3) the dural arteries and (4) the cerebral arteries at the base of the brain (see Table XX page 184) Therefore if the patient assumes an upright position when leakage of spinal fluid has occurred and the shock absorbing effect of the spinal fluid is absent or diminished the brain sags so to speak producing tension and pressure on the above mentioned structures and this action causes headache The supine position automatically reduces this tension and the headache is relieved[56, 57, 58]

Nuchal Rigidity —The patient may complain only of neck and shoulder pain or a stiff neck and such a symptom may be overlooked However in most instances when there is an occipitonuchal distribution of the pain it is severe enough that the patient complains bitterly when he assumes a vertical position This neck rigidity is what has led many to believe that meningeal irritation is present

The neck pain is probably not due in most instances to meningeal irritation but is caused

by traction on, or dilatation of, the deep lying structures (dura and blood vessels) at the base of the brain, producing stimuli which are referred via the cervical cord to the neck muscles with resulting pain and spasm [33, 57]

Visual Disturbances —These include blurring of vision difficulty in focussing colored vision confluent letters, sloping lines, and diplopia They are usually accompanied by postural headache which calls the physician's attention to the fact that the leakage of spinal fluid is responsible and steps to correct this are usually instituted However if they occur alone are unrecognized as a possible sign of decreased spinal fluid pressure and the hypotension of the spinal fluid goes untreated in a small percentage of cases a serious problem such as paralysis of the abducens nerve may result (see Chapter 24 page 227) [9]

Disturbances in the Labyrinth —Dripps and Vandam [33] in reference to hypotensive spinal headache state, "Similarly altered cerebrospinal fluid dynamics may explain the tinnitus decrease in hearing or blocking of the ears observed in 1% of our patients The anatomic connection between the cochlea and the subarachnoid space is established [60] If India ink be injected into the cerebrospinal fluid of dogs and cats, discoloration of the perilymph of the labyrinth is noted within 15 minutes Hughson [60] has demonstrated that with reduction in cerebrospinal fluid pressure there is a subsequent decrease in intralabyrinthine pressure associated with impairment of hearing for high tones The change produced in the ability of the ear to transmit high tones may be sufficient to block all appreciation of such tones In a patient recently seen by us with a severe postlumbar puncture headache and diminution in hearing both the headache and the hearing improved significantly after injection of fluid into the subarachnoid space

Dizziness occurs after spinal anesthesia and may persist for months [4, 23, 28] However its relationship to the anesthetic procedure per se is questionable In most cases it coexists with the headache of the type seen following leakage of spinal fluid [28] Arner [4] found an incidence of 0.6% dizziness in his 1,191 patients surveyed and stated, "In the present series dizziness did not occur as an isolated symptom following spinal anesthesia nor did it constitute any complication of significance following spinal anesthesia Ericsson [23] agrees with Arner that dizziness after spinal anesthesia is largely due to factors other than the block itself No specific means of prophylaxis or treatment can be prescribed for this symptom Intravenous Novocain (procaine) may be tried but in most instances it is not of much help

PROPHYLAXIS OF HEADACHE FROM HYPOTENSION OF THE SPINAL FLUID

During the past seven years at the Mason Clinic we have been using fewer and fewer spinal procedures and an ever increasing number of peripheral nerve blocks and epidural (peridural) blocks in order to avoid the possible complications of spinal anesthesia of which headache is one of the most troublesome

Both epidural and peripheral nerve blocks (such as the combination of sciatic and femoral nerve blocks for the leg and intercostal deep splanchnic [celiac plexus] block for upper abdominal surgery) have proven to be as efficacious as spinal block in our institution [39, 61, 63]

The introduction of Xylocaine (lidocaine) and Cyclaine (hexylcaine) drugs with a rapid onset of effect when given epidurally has in our estimation greatly facilitated the use of epidural block By employing these two drugs epidural anesthesia may be substituted for spinal anesthesia in most instances without sacrificing any of the advantages of spinal block

At the present time we are using only Xylocaine for epidural blocks in the thoracic or lumbar areas and Metycaine for continuous caudal blocks because of the untoward results noted from the use of Cyclaine (pages 17 and 220) To assure duration of analgesia it has been our custom to add to the Xylocaine solutions 2 mg of Pontocaine (tetracaine) per

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The neck pain is probably not due in most instances to meningeal irritation but is caused

daily fluid intake of at least 3000 cc for three days as well as 1 cc of Pitressin intramuscularly every twelve hours for three doses Concerning the Pitressin he writes, The Pitressin used in this treatment contains 10 units of pressor and 0 1 units of oxytocic activity This preparation acts on two body systems It depresses coronary blood flow to some extent, perhaps strengthens the heart contractions and induces bradycardia[67] These effects are perhaps reflex changes initiated by its peripheral vasoconstriction which causes a blood pressure rise in the anesthetized patient Hypertension is not usually observed when this drug is administered to an unanesthetized patient The second primary action of this preparation is its antidiuretic effect on the kidney The inhibition of diuresis occurs due to an increase in tubular absorption of water An antidiuretic effect will not be observed if the kidney is already excreting urine of maximum specific gravity The higher concentration of chloride accompanying its antidiuretic action may even be an increase in total salt excretion per unit of time The healthy postnatal female will not suffer a salt retention due to Pitressin The drug is not used in patients with heart disease hypertension toxemia preeclampsia or eclampsia

Size of Needle—The possibility that the gauge of the needle influences the incidence of spinal headache caused by leakage was proposed by Hoyt[48] in 1922 He sought to reduce the incidence of spinal headache by employing the "double needle" technique which essentially consists of placing a large needle into the interspinous ligament and then inserting a smaller gauge needle through it Since then this principle has been used by many foreign physicians[34,49,68,73] However until recently the use of smaller gauge needles as a means of reducing headache following dural tap has not been stressed by American authors[10,15,0,30,104,105,107]

The incidence of spinal headache is markedly reduced by the use of 22 to 27 gauge needles when compared with 20 or larger gauge needles (see Table XVII page 180 Table XXI page 186 and Table XXII, page 186)[10,15,20,30,50,74] When the hydration technique of Greene[15] is used along with the small gauge needles the incidence of spinal headache is very small (see Table XXI, page 186) Greene,[15] when evaluating his results states These findings confirm the value of reducing the diameter of the spinal needle and of hydrating the patient It is evident, however that oral hydration is not of sufficient influence to balance a leak as large as that produced by a 20 gauge needle Hydration is more effective when the needle is of 22 or 24 gauge The important factor is the diameter of the needle Even the 24 gauge needle however, requires the aid of vigorous hydration to reduce the headache to a minimal incidence Therefore, there is need for the 26 gauge size

Bevel of Needle—The bevels of most spinal needles are ground in such a fashion that they have cutting edges Greene[74,76] found from experiments that a large gauge needle with such an edge made large irregular holes in the dura while a small bore needle with a sharp point possessing rounded rather than cutting edges on the bevel separated rather than cut the longitudinal fibers permitting approximation following removal of the needle and resulting in a smaller hole (see Figure 41, page 187 and Figure 42 page 187) Thus leakage is kept to a minimum Whitacre's[77,78] pencilpoint needle functions on the same principle

A comparison between headache incidence following the use of the Greene point needle and the ordinary needle of the same gauge was not found in the literature reviewed For the past seven years at the Mason Clinic the 21 gauge Greene point needle has been used in the administration of over 3700 spinal anesthesias for all types of operations with an incidence of spinal headache of approximately 7% While we support the theory and efficacy of hydration, no specific effort to institute it was made in this series until headache occurred

Hart and Whitacre's[78] comparison of the regular beveled 20 gauge needle with the 20 gauge pencilpoint needle shows a marked re

TABLE XX

PROBABLE MECHANISM OF HEADACHE FROM LEAKAGE OF SPINAL FLUID OR MENINGITIS
(*Material from Wolff*[57])

Etiology	*Area of Head to Which Pain is Referred*
LEAKAGE OF SPINAL FLUID (Hypotension of Spinal Fluid)	
I Traction on	
A Veins passing to sagittal and transverse sinus	Front top and side of head
B Middle meningeal arteries	From eyes posterior to ears
C Intracranial portion of internal carotid arteries and main component of circle of Willis or pia arachnoid	Region of eyes or front top or sides of the head
D Basilar and vertebral arteries and branches	Back of head and neck
II Displacement of entire intracranial arterial system from right to left or vice versa	Generalized headache from eyes to neck
INFLAMMATION OF STRUCTURES AT BASE AND CONVEXITY OF BRAIN (Hypertension of Spinal Fluid)	
I Posterior fossa	Chiefly back of head
II Supratentorial fossa	Chiefly frontal or vertical

cc of Xylocaine and regardess of the volume of solution to be injected 0 2 cc Adrenalin (epinephrine) 1 1000[61] Using these techniques rather than spinal anesthesia in the majority of cases selected for regional block procedures has greatly decreased the number of headaches

However, when spinal analgesia is used the following means of reducing the incidence of spinal headache should be evaluated

Avoid Usage of the Term "SPINAL"—Due to adverse publicity given spinal anesthesia by the press, following reports of its complications at medical meetings or the awarding of substantial sums following lawsuits, most anesthesiologists who are going to give a patient a spinal anesthetic prefer to use the term "lumbar block" or "subarachnoid block Thus the patient is not alerted to the fact that spinal anesthesia is being used and consequently does not blame every ache or pain on the spinal block

Hydrate the Patient—A positive water balance on which the choroid plexus may draw to produce spinal fluid is essential[15, 70] It has been shown that adequate hydration favors regeneration of spinal fluid at a rate sufficient to replace that lost via a large dural leak while inadequate fluid intake or hypertonic dextrose solutions may aggravate the effect of even a small dural leak (see Table XXI, page 186 and Table XXII page 186)[61, 66]

Hydration should be assured prior to during and following surgery if spinal block is to be used It must be remembered that a dehydrated patient with a subnormal spinal fluid volume cannot replace the spinal fluid until the water for insensible loss and urinary excretion is provided[70] The two following hydration regimes have been proposed as effective ways of assuring a positive water balance in obstetrical patients receiving spinal blocks They are also applicable to most other patients receiving this type of anesthesia

Greene's[15] technique of a postoperative hydration regime for the obstetrical patient consists of 2 500 cc of fluid daily during the first three postpartum days and 10 units of posterior pituitary extract subcutaneously every twelve hours for four doses during the first two days postpartum If this intake cannot be maintained by fluid by mouth intravenous or subcutaneous fluids should be given He believes that using a 26 gauge needle for the spinal tap precludes the administration of pituitrin

Krueger's[70] technique of a postoperative hydration regime for postnatal patients was adapted from Greene's[15] and consists of a

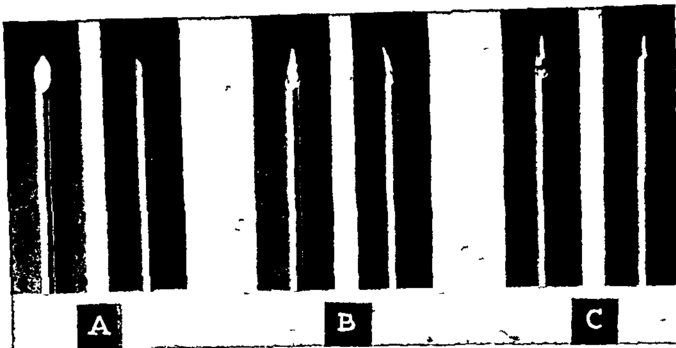


Figure 41 (A) Pitkin needle (cutting edges on bevel) (B) Greene needle (point sharp edges of bevel rounded) (C) Whitacre pencilpoint needle (point sharp bevel completely rounded)

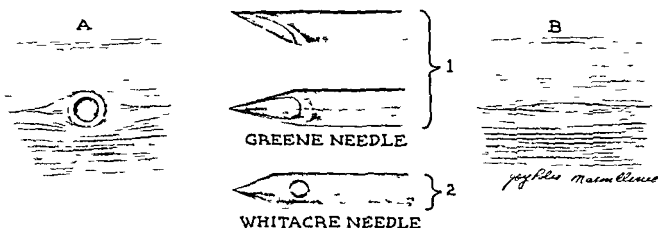


Figure 42 (A) Greene or Whitacre needle in place Only a few of the longitudinal fibers of the dura are severed most are spread—not cut (B) When needle is removed fibers tend to approximate

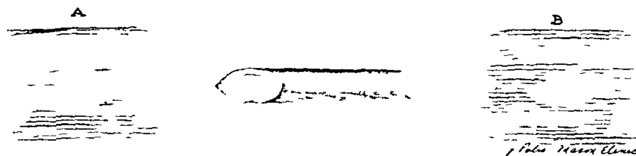


Figure 43 Crescent shaped cut in dura made by the sharp cutting edges of the bevel of the Pitkin needle which has been introduced into the dura with the bevel of the needle at right angles to the longitudinal fibers of the dura A maximum number of fibers are severed when a needle with this type of bevel is *incorrectly* inserted in this position into the subarachnoid space (B) Needle removed The hole in the dura gaps widely

TABLE XXI

REPORTED INCIDENCE OF HEADACHES FOLLOWING PUNCTURE OF THE DURA WITH NEEDLES OF VARIOUS SIZES AND POINTS

Author	*Size of Needle, Type of Point and Treatment*	*No. of Patients and Type of Operation*	*Percentage Incidence of Headache*
Greene[15]	20 Gauge without hydration	17 (obstetrical)	41.0
	20 Gauge with hydration	18	33.3
	22 Gauge without hydration	93 "	26.0
	22 Gauge with hydration	108	10.0
	24 Gauge without hydration	192	8.0
	24 Gauge with hydration	149 "	2.0
	26 Gauge with modified hydration	700 "	0.4
	(All regular beveled points on needles)		
Cann & Wycoff[10]	27 Gauge without hydration (All regular beveled points on needles)	100 (all types of operations)	5.0
Owen[80]	26 Gauge without hydration (All regular beveled points on needles)	862 (all types of operations)	0.69
Harris & Harmel[4]	24 Gauge without hydration	529 (all types)	3.59
	20 Gauge without hydration	511	8.22
	Larger needles	5 "	23.8
	(All regular beveled points on needles)		
Hart & Whitacre[78]	20 Gauge, regular point (No particular effort to hydrate)	2070 (all types)	5.0
	20 Gauge (pencilpoint) (No particular effort to hydrate)	3489	2.0

TABLE XXII

HEADACHE INCIDENCE WITH NEEDLE SIZE AND HYDRATION IN OBSTETRIC PATIENTS

(From Krueger[70])

20 pencilpoint with hydration (150 patients)	4.0%
20 pencilpoint without hydration (261 patients)	7.3%
22 beveled without hydration (30 patients)	16.6%
20 beveled with hydration (56 patients)	15.0%
20 beveled without hydration (282 patients)	21.7%

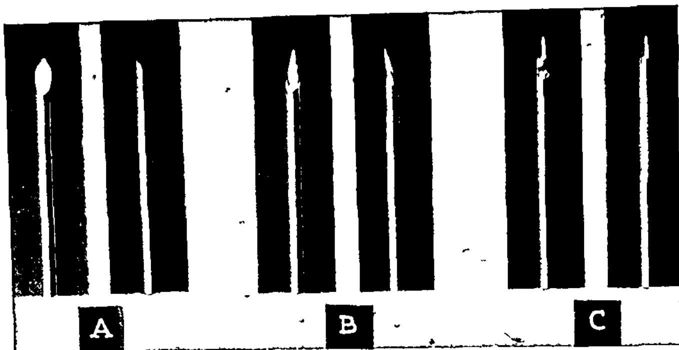


Figure 41 (A) Pitkin needle (cutting edges on bevel) (B) Greene needle (point sharp edges of bevel rounded) (C) Whitacre pencilpoint needle (point sharp bevel completely rounded)

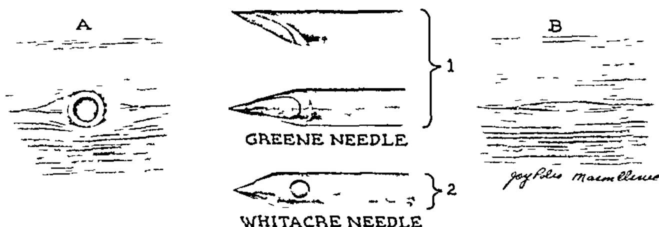


Figure 42 (A) Greene or Whitacre needle in place Only a few of the longitudinal fibers of the dura are severed most are spread—not cut (B) When needle is removed fibers tend to approximate

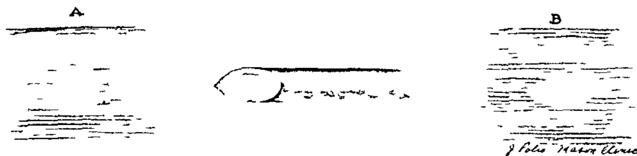


Figure 43 Crescent shaped cut in dura made by the sharp cutting edges of the bevel of the Pitkin needle which has been introduced into the dura with the bevel of the needle at right angles to the longitudinal fibers of the dura A maximum number of fibers are severed when a needle with this type of bevel is *incorrectly* inserted in this position into the subarachnoid space (B) Needle removed The hole in the dura gaps widely



Figure 44 (A) Pitkin needle in place in the subarachnoid space after being *correctly* inserted with the sharp cutting edges of its bevel parallel to the longitudinal fibers of the dura When a needle with sharp cutting edges on the bevel is inserted in this manner it cuts fewer fibers of the dura (about one half as many) than when it is inserted at right angles to the fibers (B) The hole in the dura is smaller than that produced in Figure 41 because fewer dural fibers have been severed

duction in the incidence of headache with the latter (see Table XXI page 186) Kruegers [0] study with the use of this needle in obstetrical deliveries showed the reduction in the incidence of spinal headache when it is employed with and without hydration in comparison to other gauge needles (see Table XXII page 186) Kruegers results with the 20 gauge pencilpoint needle without hydration closely approximate our results using the Greene needle

Therefore it can be concluded that the use of any needles whose bevels are non cutting will further reduce the incidence of head aches of this type

Method of Placing Needle—Most of the fibers of the dura run longitudinally i e parallel to the spinal cord [9] Because of this it has been repeatedly emphasized that if the fibers are pushed apart (this occurs automatically with a Greene or pencilpoint needle as shown in Figure 42 page 187) rather than cut across (as when an ordinary beveled needle is inserted incorrectly as shown in Figure 43 page 187) they will approximate more closely when the needle is withdrawn [1 30 34 3 74] This approximation of fibers results in less leakage of spinal fluid Therefore when a needle whose bevel has cutting edges is used the bevel should be inserted parallel to the fibers of the dura so that the needle will tend to split or spread them rather than cut them (Figure 44 page 188) This type of placement will result in a slit shaped small wound while cutting across the fibers will result in a crescent shaped hole which will tend to gape as the fibers retract (compare Figure 43 page 187 with Figure 44 page 188)

Bed Rest—If leakage is the principal cause of spinal headache then it might be reasonable to assume that the supine position without a pillow or even slight Trendelenburg position would reduce the incidence of headache Whether or not this is the case is debatable

Sicard [80] who was probably the first investigator to stress leakage as the cause of spinal headache emphasized the importance of bed rest Arner [4] supports this view Bed rest following lumbar puncture and spinal anesthesia is as pointed out in the foregoing [his study of spinal headaches] of considerable importance in the prophylactic measures against postpuncture headache In some of the cases reported above [his study of the cause of spinal headache] the headache might have been largely due to insufficient bed rest following the operation Jennings and Karabin [40] agree that bed rest markedly reduces the incidence of headache

On the other hand Underwood [81] reports an incidence of 19% incapacitating lumbar puncture headaches in his series and felt that bed rest following the tap did not reduce the

incidence Likewise Blau[17] and Levin[13] attribute little significance to bed rest

The author and his associates have not placed too much emphasis on bed rest on the basis of an unpublished study of this problem conducted at Wesley Memorial Hospital in Chicago which acted as an emergency hospital for the Navy during World War II and where a large number of emergency appendectomies were performed under spinal anesthesia on healthy young males 18 to 30 years of age Fifty of these patients were allowed to be ambulatory as soon as the effects of the spinal anesthesia were dissipated (2 to 4 hours) and an equal number were restricted to the supine position without a pillow for 24 hours No efforts to assure hydration were made The incidence of headache in the two groups showed no significant difference

Since it has been shown that a hole in the dura may not close for two weeks it is reasonable to expect that restriction to bed for only a 24 hour period would be of little help

Epidural Saline —Kaplan and Arrowood[77] have advocated the injection of saline into the epidural (peridural) space at the completion of the lumbar tap as a means of reducing spinal headache They state "After the solution containing the anesthetic drug was introduced into the subarachnoid space the needle was withdrawn into the epidural space Then 10 to 20 cc of normal saline solution was deposited before the needle was withdrawn The amount injected depended on the resistance encountered during the injection When it became difficult to introduce an additional quantity of saline solution the epidural space in that area was considered to be filled

Using this technique with a 22 gauge needle with no special point (as a Greene needle etc) they were able to reduce their incidence of headache from 7 to 1% They conclude The efficacy of this method appears to depend on the building up of a head of pressure near the dural defect and preventing leakage of spinal fluid into the epidural space This prevents a concomitant decrease in cerebrospinal fluid pressure It is not clear whether this saline solution remains in the region long enough to accomplish its mission It is conceivable that it prevents leakage long enough for a fibrin clot to form or for the parachnoid to seal the defect"

Avoid Traumatic Punctures —Multiple punctures of the dura must be avoided otherwise a great quantity of spinal fluid may be rapidly lost (fluid goes through a sieve faster than through a pipette)

Apply Epigastric Pressure —To reduce the incidence of spinal headache Leighton and Hershenson[8] advocate the use immediately postpartum of an umbilical hernia belt modified to give epigastric pressure In 404 patients studied the incidence of headache was 26% in the control group and 3 4% in those who were fitted with an umbilical hernia belt as a prophylactic measure

Administration of A C T H (corticotropin) or Antihistaminic Drugs —Ortiz and Van Der Hofstadt Alberola[108] have indicated that when headache follows spinal anesthesia it is referable to meningeal irritation even if there is hypotension of the spinal fluid They base their opinion on an increased cell count of the cerebrospinal fluid These authors advocate the use of A C T H (corticotropin) 3 to 5 mg or antihistaminic drugs namely Antistine or diethyl amino ethyl phenothiazine hydrochloride in doses of 0 1 gm and 50 mg respectively to prevent or control this increase in cell count of the cerebrospinal fluid

This type of therapy is open to question The mere performance of a puncture of the dura almost inevitably causes a reaction of the meninges as evidenced by an increased cell count of the cerebrospinal fluid (see Chapter 22 page 204) The fact that the cell count is elevated does not necessarily mean that headache will occur nor if it does that it is a specific result of meningeal irritation This increased cell count usually subsides by itself in the greater number of cases without producing headache or more specific signs of meningitis

Benadryl (diphenhydramine hydrochloride) and Dramamine (dimenhydrinate) have also been used for prophylaxis of the hypotensive type of spinal headache[103, 106] Shan

TABLE XXIII

METHODS USED AT THE MASON CLINIC TO TREAT POSTSPINAL HEADACHES FROM LOWERED SPINAL FLUID PRESSURE

I When diagnosis is made the following orders are written

A Apply abdominal binder snugly over 2 bath towels before patient sits up or is ambulatory **Rationale** Decrease epidural space increase spinal fluid pressure

B Codeine gr ½ (30 mg) and Aspirin gr 10 (600 mg) p r n for headache **Rationale** Symptomatic relief of headache

C Measured oral intake of at least 3 000 cc of water for 4 days If this is not met supplement with 5% dextrose in distilled water intravenously **Rationale** Hydration

D Nicotinamide 1¾ gr (100 mg) intramuscularly 3 times a day for 2 days **Rationale** Dilatation of choroid plexus

E Surgical pituitrin (1 2000) 1 cc intramuscularly 3 times a day for 2 days **Rationale** Antidiuretic action

N B Watch for pituitrin shock if it occurs stop pituitrin Do not give drug *to patient with* heart disease hypertension toxemia preeclampsia or eclampsia

II If severe headache persists after this treatment then the placement of a lumbar epidural (peridural) catheter and the injection of normal saline should be evaluated **Rationale** Decrease epidural space increase spinal fluid pressure

NOTE Treatment as described takes 3 days—the length of time a mild or moderate postspinal headache of this nature lasts—and therefore its efficacy may be questioned Nevertheless the patient feels an attempt to help his problem is being made

non[103] used Benadryl 50 mg by mouth preoperatively and the same dosage by mouth one and three hours postoperatively In cases where the oral route was not feasible the dosage was reduced to 20 mg and given intravenously but on the same time schedule Using the drug in this prophylactic manner he had only 5 spinal headaches in 300 cases i e 1 7% Prior to this series a control series revealed an incidence of spinal headaches of 9 4%

In the 50 obstetrical cases given Dramamine, the patient "received eight 50 mg oral doses of Dramamine in the following manner first dose after expulsion of placenta second dose four to eight hours later remaining six doses at 7 A M (5 A M for nursing mothers) and 2 P M for three consecutive days The morning doses were timed to precede arising by approximately thirty minutes" [106] The use of Dramamine reduced the incidence of headache from 38% in the 50 control cases to 8% in the 50 cases receiving Dramamine

Closing the Dural Hole with Catgut—A method of closing the hole in the dura with catgut was described by Nelson[83] and a control study on this method was conducted by Emory[84] Emory s[84] report does not show that such a procedure lowers the incidence of spinal puncture headache although Nelson[83] believed that it did

TREATMENT OF HEADACHE FROM HYPOTENSION OF THE SPINAL *FLUID*

Each physician who does a large number of lumbar punctures has his own particular method of handling the headache problem The routine at the Mason Clinic may be found in Table XXIII page 190 In general treatment of this type of spinal headache is aimed at preventing further loss of spinal fluid and elevating the spinal fluid pressure The following means of accomplishing these aims have been suggested and the fact that so many have been proposed is ample proof that no single method is entirely satisfactory It must also be remembered that most spinal headaches last only 1 to 3 days and in view of this too vigorous treatment i e retap may not be indicated unless the headache persists beyond three days

Bed Rest—As noted under prophylaxis the results obtained by bed rest are questionable There is no doubt that the supine position without a pillow relieves the headache but whether or not it shortens the course of the headache is open to question

Symptomatic Relief of Headache—A true hypotensive dural puncture headache at the onset is seldom relieved by analgesics such as salicylates On the other hand opiates i e

codeine gr ½ (30 mg) to 1 gr (65 mg) and salicylates together will control the pain and it has been our custom to give codeine gr ½ (30 mg) and Aspirin (acetylsalicylic acid) gr 10 (600 mg) as necessary (p r n) to control the headache (see Table XXIII, page 190)

Hydration—Every attempt should be made to provide a positive water balance provided this is not detrimental to the patient's general physical condition A positive water balance assures adequate fluid from which the choroid plexus can produce spinal fluid

A daily intake of 2 500 to 3 000 cc of fluid should be maintained either orally or if necessary by intravenous infusions Usually isotonic solutions i e normal saline or 5% dextrose in distilled water are used[15, 70] However the use of hypotonic solutions on the theory that these may facilitate the production of spinal fluid has been advocated[6, 8, 88] Baar[88] and Solomon[6] feel that this type of treatment definitely reduces the incidence of headache

Posterior pituitary extract or Pitressin may be used for its antidiuretic action to facilitate the storage of water as indicated on page 185 if there are no contraindications i e heart disease hypertension toxemia pre eclampsia or eclampsia It should be noted that when pituitrin or allied drugs are given so called "pituitrin shock" characterized by facial pallor nausea belching cramps and the desire to defecate may result[67] However when it does there is usually reflex slowing of the pulse and no fall in blood pressure One such case has been observed at the Mason Clinic

Abdominal Pressure—It has been shown that by applying and then releasing pressure on the abdomen a spinal headache from hypotension of the spinal fluid may be made to disappear and reappear[17] Relief obtained from abdominal pressure is probably due to compression of the inferior vena cava with consequent engorgement of the epidural and meningeal veins of the spine in the lumbar region This in turn compresses the dural sac resulting in a relative increase of vertebral cerebrospinal fluid pressure and thus the spinal fluid is forced upward to cushion the brain and prevent further shifting of the brain caudad[17, 80]

Effective abdominal pressure may be applied by placing one or two bath towels under an abdominal binder and tightening it At the Mason Clinic this method has proven useful but the pressure must be adequate However Watson[21] has tried this method on a large group of obstetrical patients with "no good results Another very effective method is to place a deflated basketball bladder under the binder and after the binder has been wrapped snugly inflating the basketball bladder

Spinal and Epidural Injection of Dextrose or Saline—*Spinal Injection*—In 1923 Jacobaeus and Frumerie[4] reported two cases of severe postspinal headache which were relieved by restoring the spinal fluid pressure to normal values with a subarachnoid injection of saline solution Since then a number of articles in which saline or dextrose or a combination of the two were injected subarachnoidally have appeared in the literature confirming these results[4, 43, 89, 90] Glesne[43] believes this method to be successful in at least 50% of the cases The amounts of 5% dextrose in normal saline which Glesne injected subarachnoidally varied from 15 to 85 cc the average being 36 cc He states "An attempt *was made to restore the* pressure to that at the initial reading [that *recorded at the time* of the original tap] *unless the amount* required proved to be excessive *under which* circumstances an approximation *within normal range* was considered adequate" In his *series* the spinal fluid pressure was read and recorded when the original lumbar tap was made Then when patients developed a spinal headache and when an attempt to correct this headache by subarachnoid injection of the dextrose and saline solution was undertaken an amount of solution necessary to approximate the original recorded spinal fluid pressure reading was injected

Epidural Injection—Rice and Dabbs[91] Risholm[9] and Denson *et al*[93] have demonstrated that similar effects may be obtained

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by epidural injections Rice and Dabbs[91] have used indwelling epidural (peridural) catheters as well as ordinary spinal needles to place the solution in the epidural (peridural) space either in the caudal or lumbar area and have found the catheter preferable Their technique is as follows The amount of saline solution injected into the peridural space varies greatly with the individual patient treated The average total dosage in our series was 82 cc Our general schedule when using the catheter method of administration consists of an initial dose of 20 cc to 30 cc The patient is then allowed to sit up and the usual result is that the headache is completely gone The patient is then rechecked in one hour Often slight discomfort will again be present at this time and an additional 30 cc is injected In a number of our cases this has been all that is required to cure the headache The catheter is left in place however taped to the patient's side and the distal end closed with a sterile Luer Lok cap On the following day the patient is examined and questioned again as to discomfort Further injections are given if needed In only the most stubborn cases was this found necessary" All twenty two of their patients with typical severe postspinal headaches had "gratifying results Again Denson *et al*[93] have found that a single epidural injection of normal saline via the caudal canal will completely relieve 84% of severe hypotensive spinal headaches and that an additional 9% receive almost complete relief Here as in the technique of Rice and Dabbs[91] the rationale for the treatment lies in elevating the epidural pressure in the sacral and/or lumbar regions which in turn decreases the volume of cerebrospinal fluid these portions of the dural sac can accommodate thereby raising the brain back onto its normal fluid cushion

The advocates of both the epidural and subarachnoid injections stress that these techniques are reserved for the *persistent severe* postspinal headache We have had occasions to evaluate these procedures at the Mason Clinic Recently three of our cases of headaches have fallen into this category and have not cleared within 2 to 5 days after their onset In all three cases the treatment was effective One case required three injections of normal saline and the others only one

It would seem that of these two types of injections epidural injections would be the more practical type since the dura would not be re punctured and a new avenue for leakage of spinal fluid would not be thus established

Dilated Choroid Plexus — The cerebrospinal fluid is formed primarily by the choroid plexus and if this plexus is dilated and a positive body water balance is maintained compensating for the loss of spinal fluid from leakage may be possible With this in mind Moyson and Lambiotte[94] suggested block of the superior cervical ganglion and others have advocated the use of the following drugs with varying success (1) caffeine with sodium benzoate[81] (2) ergotamine tartrate[3, 95] (3) dihydroergotamine[96] (4) intravenous alcohol[97] (5) nicotinamide[98] (6) Cafergot (ergotamine tartrate and caffeine alkaloid)[98] (7) Invocain (strychnine)[99] (8) desoxycorticosterone acetate[100, 101] and (9) Romicol tartrate (B pyridyl carbenol tartrate)[102]

It must be remembered that as yet no one drug has been found successful in all cases

Psychotherapy —In addition to the specific therapy listed above the psychogenic factor must not be overlooked Hart and Whitacre[78] state "In addition to definitive treatment the psychogenic factor should be given attention When such a factor is unmistakably present psychotherapy may prove of benefit It has been helpful to prevail on doctors nurses and all others who come in contact with the patient not to draw attention to the possibility that the patient's headache is a complication of spinal anesthesia The patient who remains unaware of the existence of this complication is much less likely to become one of its victims"

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CHAPTER 21

Backache

BACKACHE following a regional block particularly spinal block is neither a serious nor disabling complication but it can be annoying to the patient the surgeon and the anesthetist Since very little concern is normally exhibited by the physician when back pain occurs from block procedures where a spinal or epidural tap has not been attempted, this chapter will deal mainly with back pain which follows spinal and epidural anesthesia The incidence of back pain following spinal anesthesia varies from between 2 to 25% depending on the type of investigation conducted[1-6]

ETIOLOGY

Back pain may occur following any type of anesthesia and its frequency after regional block procedures is probably no greater than after general anesthesia[7,8] Nevertheless, when it occurs following spinal anesthesia it is inevitably blamed on this procedure by both the physician and the patient irrespective of whether or not it occurs at the site of the spinal tap or in an unrelated area In most of these cases the surgeon particularly when he has not administered the anesthetic accepts this assumption as fact and makes no attempt to establish the source of the pain more accurately by physical examination or by asking (1) was the pain present prior to the anesthetic? (2) is it caused by arthritic changes? (3) is it the result of metastatic carcinoma? (4) is there x ray evidence of changes at the site of the spinal tap? and (5) what psychological factors if any are involved? It is interesting to note that Arner[9] after studying 1 191 patients 603 of which were surveyed by questionnaires and 588 by clinical examination and roentgen studies found that a total of 233 patients (19 6%) had back pain but he states only in 35 cases (3 0%) was any relation between operation and back pain possible or probable”

In many instances the *exact* etiology of back pain attributable to spinal or epidural anesthesia is unknown but the following have been proposed

Relaxation of the Back Musculature—A number of authors mention this as a possible cause of back pain and anatomically it is a plausible explanation[3,5,6] Normally, the back curves in the lumbar area and in many patients this curve is accentuated i e the patient is lordotic When patients, particularly those with lordosis are made to lie supine for any period of time without adequate support for their normal lumbar curve pain may result Any patient who is confined to bed for any lengthy period of time will usually complain of backache either during the time he is in bed or when he first becomes ambulatory whether he has had an anesthetic or not If he is not only subjected to a supine position for a time but the greater majority of his back muscles are relaxed by a regional block procedure e g a spinal or epidural anesthesia the lumbar curve its muscle support removed automatically flattens because of gravity (see Figure 45 page 198) The effect of this flattening is to stretch the muscles ligaments and joint capsules of the spine so that when motor and sensory function return the lumbar area becomes painful Therefore since this pain is usually limited to the lumbar area and since this is the area used most frequently

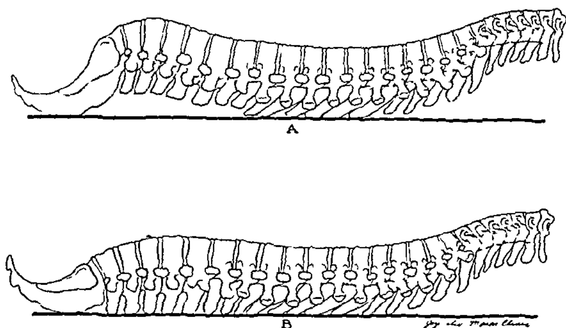


Figure 45 (A) Normal lumbar curve (B) Lumbar curve flattened or straightened as a result of the paralysis of the sensory and motor nerves of the dermatomes and consequent relaxation of the muscles of the vertebral column

for a tap the patient blames the pain on the anesthetic procedure

Trauma to Anatomical Structures.—Trauma to the intervertebral ligaments periosteum of the vertebra the annulus fibrosus (intervertebral disc) the blood vessels in these structures or subcutaneous tissues or to a combination of the above has been cited as another cause of backache [7, 8, 9, 10, 11, 12]

It has been shown that if hypertonic saline is injected into the spinal ligament periosteum or back muscles a deep aching pain from this mechanical pressure results [13] Therefore, it seems reasonable to expect that hemorrhage into a ligament caused by a dull needle or multiple explorations regardless of the gauge of the needle may produce the same type of pain

Damage to the intervertebral disc by trauma from the spinal needle has been proposed by Pease [10] and Dripps and Vandam [11] as a possible cause of back pain following spinal tap Wiberg,[14] while performing back operations under local anesthesia has caused lumbosacral pain by pressing on the external surface of the annulus fibrosus, and Lindblom [15] during discography has noted pain as the needle is inserted into the disc for this procedure However, in a personal communication to Amer,[7] Lindblom notes that over 500 disc punctures have been performed using Diodrast (iodopyracet) solutions without any immediately noted contraindications Nevertheless a number of reports have appeared in the medical literature citing disc lesions following spinal punctures [16, 17, 18, 19]

Trauma from Positioning the Patient.—Following the establishment of spinal anesthesia with paralysis of both the sensory and *motor nerve fibers it is not unusual for the* patient to be subjected to what might be termed "rough handling" In other words painful stretching of the muscles and ligaments may occur in the anesthetized area without complaint from the patient or con*traction of the muscles to prevent this trauma* This is particularly true in the patient who is to undergo a procedure for which the lithotomy position is indicated (a position which in itself tends to straighten the lumbar curve) Seldom are both legs raised and lowered at the same time and there is usually little concern as to whether or not the legs are too widely abducted or flexed It is not unusual for these patients to complain bitterly in the postoperative period of low back pain with

radiation to the thighs and calves of the legs It has been concluded at the Mason Clinic that much of the back pain which occurs, particularly that which follows the lithotomy position can be attributed to faulty positioning of the patient for back pain will appear in these patients whether a general or spinal anesthetic is administered

Trauma from the Operative Procedure.—A retractor misplaced during a pelvic operation or a difficult forceps extraction during a vaginal delivery may traumatize a nerve resulting in low back pain with sciatic radiation

Infection.—Reports of infected intervertebral discs epidural abscess and/or osteomyelitis have appeared and it is obvious that such conditions would result in back pain [9, 21]

Psychological Aspects.—At the Mason Clinic epidural (peridural) blocks are performed in preference to spinal (subarachnoid) block wherever possible and the patient is pointedly informed that he is not receiving a spinal anesthesia In these cases the complaint of back pain has been conspicuous by its infrequency although trauma to the ligaments etc during the placement of the needle in the epidural space must occur with the same frequency as during spinal administrations *Thus the question arises If trauma* from the needle to the subcutaneous tissues spinal ligaments periosteum of the bone and/or muscles is to be considered as a prime cause of low back pain following spinal block why is the incidence of back pain following epidural block not as great as after spinal block? Clearly, the needle on its way to the epidural space must traverse these anatomical structures in the same fashion as in spinal block and in most instances the needles used for epidural blocks are larger i e 19 gauge in contrast to 21 gauge or smaller

This question is not proposed to imply that trauma to these structures cannot produce backache or that back pain after either of these types of anesthesia is not real Nevertheless it presents an intriguing and baffling aspect of the problem and calls for further study

SIGNS AND SYMPTOMS

The signs and symptoms of back pain following spinal or epidural anesthesia are

Superficial or Deep Pain.—If the pain is caused by the puncture it usually does not have sciatic nerve distribution When sciatic nerve distribution is present, it usually indicates trauma from the positioning of the patient or from the operation

Muscle Spasm.—If the pain is severe the muscles in the lumbar area may be felt by the examining physician to be in spasm and the patient will in all probability limit his motion i e, "splinting," occurs The muscle spasm may be either bilateral or unilateral

X-ray.—*Usually the roentgen studies of the* vertebral column are negative However in some instances myelography or discography may reveal organic changes which may or may not have been caused by the regional block procedure [2]

PROPHYLAXIS

As has been noted in other chapters concerned with spinal anesthesia an attempt is made at the Mason Clinic to use other regional block procedures, i e, epidural block, sciatic and femoral nerve block, intercostal celiac plexus block etc, whenever possible in preference to spinal anesthesia This preference is not based on a fear that the serious complications of spinal anesthesia, i e, cauda equina syndrome, etc may result, but rather to avoid the frequent and less serious but annoying complications of spinal anesthesia of which backache and headache are probably the most prominent When spinal or epidural anesthesia is employed, one or more of the following precautions may help to avoid back pain

Maintain the Normal Lumbar Curve.—The normal lumbar curve should be maintained by adequate back support while the patient is on the operating table as well as in bed after the operation until the block has dissipated itself This is necessary since spinal and epidural anesthesia cause the back to lose most of its muscular support during the

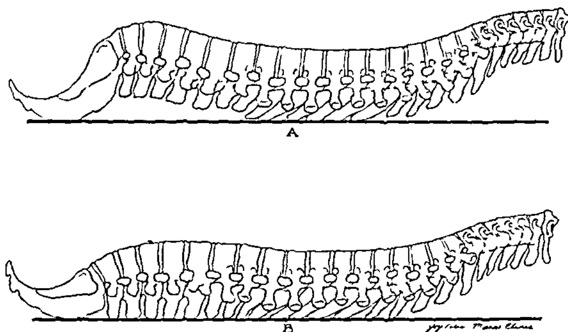


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time the local anesthetic agent is effective Carefully supporting the back of the lordotic patient is of particular importance

Avoid Traumatizing Tissues—Every effort should be made to avoid multiple punctures—if a tap cannot be executed after two or three attempts another type of anesthesia should be considered This is singularly true if the patient becomes restless uncooperative or if he objects to the continuation of the procedure If the block is administered under these conditions the patient will blame any subsequent pain or complication on the procedure regardless of whether or not there is an association and if a spinal block is executed under protest the court may uphold a charge of "technical assault"[3]

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When other positions are to be used i e lateral decubitus prone etc the patient should be moved slowly with adequate help—he must not be roughly tugged into position

Avoid Infection—Since any break in technique may result in infection abscess and other processes which usually cause pain asepsis must be assured (see Chapter 22 page 206)

Do Not Use the Word "Spinal"—When discussing a spinal anesthesia with a patient the physician should use terms such as lumbar block" or "subarachnoid block" Many patients will blame every little ache or pain on a spinal block because friends have complained following this type of anesthesia or they have read of its untoward effects in a magazine or newspaper The hospital personnel should also be warned against the use of the term "spinal

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If the pain is superficial it will usually pass away in a day or two and "skillful neglect with watchful waiting will suffice However if a hematoma has formed in the tissue heat, physiotherapy and the control of the pain with large doses of acetylsalicylic acid (Aspirin),* Tolserol, etc and/or an opiate may be necessary Infection should receive the appropriate therapy i e antibiotics drainage etc

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CHAPTER 22

Meningitis and Meningeal Irritation

Meningitis is a serious complication of regional block procedures. It may occur following a lumbar puncture alone, a spinal anesthesia, a paravertebral, or an epidural (peridural) block.[1,18,49,50] The patient who survives a meningitis may be disabled by its sequelae. Fortunately, present day means of sterilization and the decreased toxicity of local anesthetic agents have made both septic and aseptic meningitis from a regional block procedure relatively rare.

Meningeal Irritation.—The terms "meningeal irritation" and "meningismus" may be used interchangeably to indicate a signs and symptom complex which includes: (1) cervical rigidity, (2) a positive Kernig's sign, (3) nausea and vomiting, and (4) an increased pressure of the cerebrospinal fluid. Meningeal irritation brings about no significant pathological changes in the spinal fluid other than the increased pressure. It may herald the onset of an aseptic or septic meningitis, but it also may accompany diseases such as pneumonia and never progress to meningitis.

Meningitis.—This is an inflammation of the three membranes that envelop the brain and spinal cord, i.e., the dura mater, pia mater and arachnoid.[19] It is normally characterized by general malaise, headache, fever, head retraction and positive cerebrospinal fluid findings. It is a specific disease entity and may be classified as either septic or aseptic.

"*Septic meningitis*" is a term used to indicate a meningitis in which bacteria can be detected in the spinal fluid.[20,21]

"*Aseptic meningitis*" is a term used to denote an inflammatory condition in which all the signs and symptoms of a meningitis are present except that bacteria cannot be found in the smears or culture of spinal fluid.[20,21]

It is unfortunate that the term "meningismus" is often erroneously used synonymously with the term "aseptic meningitis." Meningismus is a sign and symptom complex and is only a part of the clinical picture of aseptic meningitis, the diagnosis of which must be established by examination of the cerebrospinal fluid. To avoid any misunderstanding, therefore, the term "meningeal irritation" is preferred in this text to its synonym "meningismus."

ETIOLOGY

Whenever the epidural or subarachnoid spaces are entered, a meningeal irritation may ensue which may or may not progress to an aseptic or septic meningitis.

Meningeal Irritation.—The specific causes of meningeal irritation alone, following a regional *block procedure in a patient with no* infectious disease are usually unknown. In all probability they are the same as those which produce an aseptic meningitis, but the stimulus initiating the response and the resulting inflammatory reaction are less severe and subside more rapidly.

On the other hand, if a disease process, e.g., a pneumonia, is present at the time a block is executed, one might reasonably assume that an ensuing meningeal irritation is due to that disease rather than to the block. Nevertheless, the possibility that the anesthetic technique was responsible cannot be discounted.

Aseptic Meningitis.—Aseptic meningitis may be caused by one or more of the following: (1) the local anesthetic agent, (2) indwelling spinal catheters or needle, (3) hemorrhage, (4) puncture of the dura, and (5) diagnostic procedures and/or the surgical procedure.

Local Anesthetic Agents.—Formerly aseptic meningitis from the use of drugs such as Spinocain (strychnine sulfate, Novocain, starch paste, alcohol and normal saline), Tropacocaine (benzoyl tropein), and Stovaine (amylocaine), etc. was not uncommon. Today, with refinements in synthesis and improvements in packaging, the time-tested local anesthetic agents, i.e., Pontocaine, Novocain, Metycaine, Intracaine, and Nupercaine, usually do not cause chemical irritation of the meninges provided they are used in the dosage usually recommended. Nevertheless, in a very small number of patients the nerve tissue may be markedly irritated by the local agent. Merritt and Fremont-Smith[2] point out that the injection of any foreign substance into the subarachnoid space will result in some form of irritation of the meninges. Reaction to the time-tested local anesthetic agents is usually minimal, but at rare times the nerve tissue may show hyperergy, i.e., the tissue reacts in a normal fashion but overreacts (see Chapter 1, page 8). Under such circumstances an aseptic meningitis may result.

Indwelling Spinal Catheters.—In most instances plastic and ureteral indwelling catheters have replaced the use of indwelling needles for continuous spinal (subarachnoid) and epidural (peridural) anesthesia in those instances where prolonged periods of analgesia are required. Their safety as well as nonirritant qualities have been shown.[3] *Nevertheless* their mere presence as a foreign body in either the subarachnoid or epidural space may result in mechanical irritation and inflammation.

In three cases at the Mason Clinic where plastic catheters were left in the epidural space following surgery for the purpose of effecting pain relief in the postoperative period, the signs and symptoms of meningeal irritation developed 12 to 24 hours following surgery. The catheters used and the equipment necessary to place them had been heat sterilized. Injections of 7 cubic centimeters of a 2% Xylocaine (lidocaine) solution without Adrenalin were administered through these catheters as necessary during the postoperative period to keep the patients free of pain from the operation. Relief of the signs and symptoms of meningeal irritation occurred in all cases within 12 hours after the removal of the catheters.

The following two questions arose concerning these three cases: what caused the irritation and what specific form of irritation resulted?

In these cases the meningeal irritation could have been caused by either the local anesthetic agent, as was suggested by Cleland,[24] or by the plastic catheters. It is our belief that either the catheter or some unknown cause was responsible. This belief is substantiated by the fact that my associates and I have employed 2% Xylocaine (lidocaine) for single dose lumbar epidural analgesia as well as continuous caudal (malleable needle technique) in over 1700 cases without causing meningeal irritation. Other physicians have found Xylocaine (lidocaine) to be nonirritating when not contaminated by metal ions, etc.[26,27]

Since these patients improved rapidly following the removal of the plastic catheters, there was no indication for a diagnostic spinal tap and it was not determined whether or not a meningitis had ensued. The rapid disappearance of signs and symptoms would probably indicate a meningeal irritation alone.

Hemorrhage.—Subarachnoid bleeding may occur following lumbar puncture from (1) spontaneous rupture of a cerebral or spinal cord vessel,[28] (2) alterations in cerebrospinal fluid pressure, or (3) trauma to vessels from the placement of the needle.[29-31] If the patient has a blood dyscrasia or is receiving anticoagulant therapy at the time the bleeding starts, the situation is doubly precarious. Jackson[30] has shown that if blood gains entrance to the subarachnoid space, signs of

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the same, those due only to simple meningeal irritation resolve in 2 to 3 days and usually do not become progressively worse The general malaise the headache and occasionally slight head retraction—signs common to both complications—lead the physician to sample the spinal fluid for he is fearful of a meningitis and wishes to establish a definite diagnosis *The outstanding method of differentiating between simple meningeal irritation and the two types of meningitis is the laboratory analysis of the cerebrospinal fluid* This is the only way of settling the diagnosis other than further waiting

In general septic or aseptic meningitis will produce the following signs and symptoms with the exception of spinal fluid findings However in septic meningitis their onset may be more rapid and the course may be more fulminant

Headache —This usually signals the onset of either a simple meningeal irritation or a meningitis At first it is mild but increases in severity and possesses a "bursting" character as meningitis develops[10] *It is present in all positions and at all times and is associated with a hypertension of the cerebrospinal fluid which differentiates it from the usual headache associated with hypotension of the spinal fluid* According to Brain[10] "It may be diffuse or mainly frontal and usually radiates down the neck and into the back being associated with pain in the spine which radiates to the limbs especially to the lower limbs

While a high spinal fluid pressure is associated with this type of headache it may not be the cause of it Macintosh[11] states "It is extremely doubtful whether mere rise of cerebrospinal fluid pressure does cause headache Symonds reports a case in which saline was injected intrathecally and the patient s normal pressure of 170 mm water was raised to and maintained at 550 mm water for some minutes without causing symptoms and there are pathological states which result in similar high pressures yet do not cause headache"

Fever —The temperature varies between 100 and 102 F but in acute cases of meningitis may reach 104 F or 105 F

Pulse —The pulse may be normal or slow, i e, 50 to 60 early in the disease When the pulse starts to increase above its normal rate the condition is becoming more severe and the onset of an acute meningitis is indicated

Respirations —There is usually only a slight increase in respirations but if septic meningitis results and is not controlled by antibiotics, various forms of respiratory irregularity may be seen

Vomiting.—This is not an unusual occurrence

Convulsions —These seldom occur in the adult patient but are not infrequent in the child

Special Signs of Irritation of the Meninges —Cervical rigidity, head retraction Kernig s sign (when lying supine with the thigh flexed upon the abdomen the leg cannot be completely extended[10]), and Brudzinski s sign (identical contralateral reflex reciprocal contralateral reflex and neck sign[10]) are usually present

Cerebrospinal Fluid Findings —As mentioned previously, the composition of the spinal fluid is the deciding factor in determining whether an aseptic or septic meningitis or only a simple meningeal irritation is present (see Table XXIV page 206)

Meningeal Irritation —When meningeal irritation alone occurs the spinal fluid pressure is increased but the cell count is normal or only slightly elevated (50 to 100 or less per cu mm) and the fluid is clear The glucose proteins and chlorides are not increased but in some instances the latter two may be slightly decreased No bacteria are found

Aseptic Meningitis —In aseptic meningitis there is an increase in the cell content of the spinal fluid usually from 100 to 1500 per cubic millimeter although in unusual cases it may be higher Reynolds and Wilson[12] reported three cases following lumbar puncture which showed signs of meningeal irritation and in which repeated taps revealed "sterile cloudy spinal fluid with cell counts ranging from 2 900 to 17 670" The fluid depending on the number of cells may be clear or turbid Both polymorphonuclear and mononuclear

aseptic meningitis may occur—he terms this condition aseptic hemogenic meningitis At the Mason Clinic we have seen one case of severe spontaneous subarachnoid hemorrhage which exhibited all the signs and symptoms of an acute meningitis It occurred on the third postoperative day following an intercostal splanchnic (celiac) block for a gastric resection The diagnosis was established by a spinal tap Although this particular case was not associated with the anesthetic procedure it does substantiate the fact that subdural bleeding can produce the signs of acute aseptic meningitis[23]

Puncture of the Dura—Even a diagnostic lumbar puncture may produce an increase in the normal cell count of the spinal fluid to 50 or 100 cells and produce the signs of meningeal irritation However aseptic meningitis seldom results from simple dural puncture if properly sterilized needles have been used unless blood vessels have been injured too

Diagnostic Procedures and/or the Surgical Procedure—It is not unusual for a myelography to be performed prior to lumbar disc surgery and a spinal anesthesia may be used as the anesthesia of choice in these patients An aseptic meningitis may be produced by the mechanics of either of these procedures or by the drugs injected Moreover if in addition to the above the dura is traumatized during the operation the possibility of another etiological factor has been introduced The only proven cases of aseptic meningitis which my associates and I have seen occurred in this kind of case

The patient was a 36 year old white female who was suspected of having a ruptured intervertebral disc A myelogram with Pantopaque (ethyl iodophenylundecylate) three days prior to surgery confirmed the diagnosis The Pantopaque was recovered except for a "few droplets of oil" The Pontocaine spinal anesthesia and surgery were uneventful However on the second postoperative day the patient began to complain of a headache which persisted in all positions On the third postoperative day her fever rose from 100 F to 103 F she showed signs of cervical rigidity and her pulse rate was 120 Spinal tap revealed (1) increased pressure (2) no red blood cells (3) 1210 white blood cells 79% polymorphonuclear cells and 21% lymphocytes (4) the smear was negative except for "amorphous debris and many white cells" (5) the 24 hour and 72 hour cultures were negative (6) the chlorides and glucose determinations were normal and (7) the protein content was elevated

Immediately after this diagnostic spinal tap the patient had been placed on Terramycin therapy a broad spectrum antibiotic so that if the cultures proved to be positive the drug therapy might control the infection more rapidly On the fourth postoperative day her temperature rose to 105 and then slowly subsided and read 99 6 F on the eighth postoperative day She was discharged from the hospital on the twelfth postoperative day with no evidence of neurologic residua

The exact cause of the aseptic meningitis is difficult to determine in this case but it could have been the diagnostic myelogram the anesthetic procedure itself the surgical trauma to the dura or a combination of these

Septic Meningitis—The incidence of meningitis varies widely Thorsen[7] estimated its incidence in 1 in 800 cases and Arner[10] reported one case in 21 230 The incidence is of little significance except to point out that this very serious complication does occur Septic meningitis is caused by the introduction of bacteria into the subarachnoid space This may occur because of (1) *faulty technique in sterilization of instruments and ampules* (2) *a break in technique during the placement of the needle* (3) *trauma* or (4) *the execution of a spinal tap in a patient with a septic condition particularly bacteremia*

Cases of osteitis or epidural abscess following a spinal tap have been noted[23, 3, 31] Either of these may be followed by a septic meningitis

SIGNS AND SYMPTOMS

While the early signs and symptoms of meningeal irritation and meningitis are much

the same, those due only to simple meningeal irritation resolve in 2 to 3 days and usually do not become progressively worse. The general malaise, the headache, and occasionally slight head retraction—signs common to both complications—lead the physician to sample the spinal fluid, for he is fearful of a meningitis and wishes to establish a definite diagnosis. *The outstanding method of differentiating between simple meningeal irritation and the two types of meningitis is the laboratory analysis of the cerebrospinal fluid.* This is the only way of settling the diagnosis other than further waiting.

In general, septic or aseptic meningitis will produce the following signs and symptoms, with the exception of spinal fluid findings. However, in septic meningitis their onset may be more rapid and the course may be more fulminant.

Headache.—This usually signals the onset of either a simple meningeal irritation or a meningitis. At first it is mild but increases in severity and possesses a "bursting" character as meningitis develops.[20] *It is present in all positions and at all times, and is associated with a hypertension of the cerebrospinal fluid, which differentiates it from the usual headache associated with hypotension of the spinal fluid.* According to Brain,[20] "It may be diffuse or mainly frontal, and usually radiates down the neck and into the back, being associated with pain in the spine which radiates to the limbs, especially to the lower limbs."

While a high spinal fluid pressure is associated with this type of headache, it may not be the cause of it. Macintosh[11] states: "It is extremely doubtful whether mere rise of cerebrospinal fluid pressure does cause headache. Symonds reports a case in which saline was injected intrathecally and the patient's normal pressure of 170 mm. water was raised to and maintained at 550 mm. water for some minutes without causing symptoms . . . and there are pathological states which result in similar high pressures yet do not cause headache."

Fever.—The temperature varies between 100° and 102° F., but in acute cases of meningitis may reach 104° F. or 105° F.

Pulse.—The pulse may be normal or slow, i.e., 50 to 60, early in the disease. When the pulse starts to increase above its normal rate, the condition is becoming more severe and the onset of an acute meningitis is indicated.

Respirations.—There is usually only a slight increase in respirations, but if septic meningitis results and is not controlled by antibiotics, various forms of respiratory irregularity may be seen.

Vomiting.—This is not an unusual occurrence.

Convulsions.—These seldom occur in the adult patient but are not infrequent in the child.

Special Signs of Irritation of the Meninges.—Cervical rigidity, head retraction, Kernig's sign (when lying supine with the thigh flexed upon the abdomen the leg cannot be completely extended[19]), and Brudzinski's sign (identical contralateral reflex, reciprocal contralateral reflex, and neck sign[19]) are usually present.

Cerebrospinal Fluid Findings.—As mentioned previously, the composition of the spinal fluid is the deciding factor in determining whether an aseptic or septic meningitis or only a simple meningeal irritation is present (see Table XXIV, page 206).

Meningeal Irritation.—When meningeal irritation alone occurs, the spinal fluid pressure is increased but the cell count is normal or only slightly elevated (50 to 100 or less per cu. mm.) and the fluid is clear. The glucose, proteins, and chlorides are not increased, but in some instances the latter two may be slightly decreased. No bacteria are found.

Aseptic Meningitis.—In aseptic meningitis there is an increase in the cell content of the spinal fluid, usually from 100 to 1500 per cubic millimeter, although in unusual cases it may be higher. Reynolds and Wilson[17] reported three cases following lumbar puncture which showed signs of meningeal irritation and in which repeated taps revealed sterile cloudy spinal fluid with cell counts ranging from 2,900 to 17,670. The fluid, depending on the number of cells, may be clear or turbid. Both polymorphonuclear and mononuclear

aseptic meningitis may occur—he terms this condition 'aseptic hemogenic meningitis.' At the Mason Clinic we have seen one case of severe spontaneous subarachnoid hemorrhage which exhibited all the signs and symptoms of an acute meningitis. It occurred on the third postoperative day following an intercostal splanchnic (celiac) block for a gastric resection. The diagnosis was established by a spinal tap. Although this particular case was not associated with the anesthetic procedure, it does substantiate the fact that subdural bleeding can produce the signs of acute aseptic meningitis.[48]

Puncture of the Dura.—Even a diagnostic lumbar puncture may produce an increase in the normal cell count of the spinal fluid to 50 or 100 cells and produce the signs of meningeal irritation. However, aseptic meningitis seldom results from simple dural puncture if properly sterilized needles have been used, unless blood vessels have been injured too.

Diagnostic Procedures and/or the Surgical Procedure.—It is not unusual for a myelography to be performed prior to lumbar disc surgery and a spinal anesthesia may be used as the anesthesia of choice in these patients. An aseptic meningitis may be produced by the mechanics of either of these procedures or by the drugs injected. Moreover, if in addition to the above the dura is traumatized during the operation, the possibility of another etiological factor has been introduced. The only proven cases of aseptic meningitis which my associates and I have seen occurred in this kind of case.

The patient was a 36 year old white female who was suspected of having a ruptured intervertebral disc. A myelogram with Pantopaque (ethyl iodophenylundecylate) three days prior to surgery confirmed the diagnosis. The Pantopaque was recovered except for a "few droplets of oil." The Pontocaine spinal anesthesia and surgery were uneventful. However, on the second postoperative day the patient began to complain of a headache which persisted in all positions. On the third postoperative day her fever rose from 100 F. to 103 F., she showed signs of cervical rigidity and her pulse rate was 120. Spinal tap revealed (1) increased pressure, (2) no red blood cells, (3) 1210 white blood cells 79% polymorphonuclear cells and 21% lymphocytes, (4) the smear was negative except for "amorphous debris and many white cells," (5) the 24 hour and 72 hour cultures were negative, (6) the chlorides and glucose determinations were normal and (7) the protein content was elevated.

Immediately after this diagnostic spinal tap the patient had been placed on Terramycin therapy, a broad spectrum antibiotic, so that if the cultures proved to be positive the drug therapy might control the infection more rapidly. On the fourth postoperative day her temperature rose to 105 and then slowly subsided and read 99.6 F. on the eighth postoperative day. She was discharged from the hospital on the twelfth postoperative day with no evidence of neurologic residua.

The exact cause of the aseptic meningitis is difficult to determine in this case but it could have been the diagnostic myelogram, the anesthetic procedure itself, the surgical trauma to the dura, or a combination of these.

Septic Meningitis.—The incidence of meningitis varies widely. Thorsén[7] estimated its incidence in 1 in 800 cases and Arner[10] reported one case in 21,230. The incidence is of little significance except to point out that this very serious complication does occur. Septic meningitis is caused by the introduction of bacteria into the subarachnoid space. This may occur because of: (1) *faulty technique in sterilization of instruments and ampules,* (2) *a break in technique during the placement of the needle,* (3) *trauma,* or (4) *the execution of a spinal tap in a patient with a septic condition, particularly bacteremia.*

Cases of osteitis or epidural abscess following a spinal tap have been noted.[43, 45, 61] Either of these may be followed by a septic meningitis.

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TABLE XXIV

COMPARISON OF TYPICAL SPINAL FLUID FINDINGS IN MENINGEAL IRRITATION ASEPTIC MENINGITIS AND SEPTIC MENINGITIS

	Normal Spinal Fluid	*Meningeal Irritation*	*Aseptic* Meningitis*	*Septic* Meningitis*
Pressure	110 mm H_2O	Elevated	Elevated	Elevated
Color	Clear	Clear	Clear to Xanthochromic	Xanthochromic
Cell Count	2–10 per cu mm lymphocytes or mononuclear cells	50–100 per cu mm both polymorphs and mononuclear	50–1500 per cu mm, predominantly lymphocytes but at times many polymorphs	Usually over 1500 per cu mm, predominantly polymorphonuclear
Protein	15–40 mg	Normal or slightly lowered	Slightly increased	Increased
Glucose	50–80 mg per 100 cc	Normal	Normal	Minimal or absent
Chloride	725–750 mg per 100 cc	Normal or slightly decreased	Normal	Reduced to under 700 mg per 100 cc
Presence of bacteria in smear and/or culture	None	None	None	Present

* If diagnostic spinal tap done early in course of disease typical picture of an aseptic or septic meningitis as given here may not be seen

cells are present but usually the latter predominate The protein content is somewhat elevated The glucose and chlorides are usually normal The pressure is markedly elevated and colloidal gold test shows a meningitic curve No bacteria are found

Septic Meningitis—In septic meningitis the condition of the spinal fluid ranges from a slight turbidity to a definitely purulent appearance depending on the number of leukocytes present The spinal fluid pressure is increased as is the total protein The cells are predominantly polymorphonuclear and may be present in large numbers (thousands per cubic millimeter) The chlorides are reduced to approximately 650 mg per 100 cc Glucose is minimal or absent and the colloidal gold test shows a meningitic curve Bacteria may be seen on smears or may be isolated by culture

Miscellaneous Signs —These may occur when either an aseptic or septic meningitis develops but are usually absent when only simple meningeal irritation occurs (1) delirium drowsiness stupor and coma (2) photophobia (3) ptosis diplopia and squint (4) sluggish tendon reflexes (5) facial paresis and (6) irregular pupils

PROPHYLAXIS

The prophylaxis of aseptic or septic meningitis and simple meningeal irritation is the same and consists of the following

Adequate Sterilization of Equipment —All the instruments to be used should be carefully and adequately cleaned and sterilized To date we have not had a septic meningitis although we perform over 3 000 regional block procedures per year of which approximately 2 000 are either spinal or epidural blocks Our routine technique of cleaning and sterilizing the equipment consists of

Cleaning — A number of detergent products, e g , Haemo Sol, Detergex etc for cleaning needles and syringes are available but we believe that there is less risk if soap and water or water only are used for reports show that detergent cleansers have been responsible for cord degeneration[36] The following steps are meticulously performed

I The needles and syringes are washed with copious amounts of clear tap water

II A rinse with ether follows

III The needle hubs are swabbed out with an applicator dipped in ether

Sterilization —Heat sterilization alone is above reproach if a law suit should develop (see Chapter 23 page 221)

I A sterilizer control (Diack Propper Sterilex etc) is placed in each tray as it is wrapped

II The tray is then autoclaved at 255 to 260 Fahrenheit under 18 to 22 pounds of pressure for 30 minutes

III The pressure is released from the autoclave and the steam evacuated The tray is dried by creating a vacuum in the sterilizer for 10 minutes following which the sterilizer door is opened and the tray is left in the opened autoclave for an additional 10 minutes

IV Once the tray is dry it is stored on a shelf away from all solutions This point is stressed for if solutions are unsuspectedly spilled on the tray it may become contaminated even though the sterilizer control in the tray shows adequate sterilization when it is checked

V When the tray is opened for use the sterilizer control is examined to assure adequate sterilization This precaution serves as a valuable check on the sterilizer and the personnel operating it

Adequate Sterilization of Local Anesthetic Agents and/or Solutions —Every attempt should be made to assure sterility of the anesthetic solution *The author never uses hospital prepared solutions* for any regional block procedure All drugs and equipment used in these procedures are heat sterilized The drugs and solvents used in spinal and epidural blocks at the Mason Clinic are Metycaine solutions Pontocaine crystals Novocain crystals Xylocaine solutions Adrenalin solutions, ephedrine hydrochloride in solution distilled water normal saline and 10% dextrose solutions

The multiple dose bottles of Metycaine 1.5% (250 cc size) are purchased from the pharmaceutical house This drug is employed in the obstetrical section for caudal anesthesia only The Pontocaine and Novocain solutions are prepared from crystals immediately prior to the regional block procedure, not by the hospital but by the physician who is to perform the block The crystals are received in small supposedly sterile single-dose ampules When spinal fluid is not used the solvent to dissolve these crystals for spinal anesthetics is obtained from 5 cc ampules of distilled water (Abbott) The 2% aqueous Xylocaine solutions without Adrenalin which we use for all epidural blocks in surgery are procured in the 20 cc vials rather than the multiple dose 50 cc vials so that they may be used only once Saline to dilute the 2% Xylocaine to a 1% solution where a volume of more than 20 cc is to be used is purchased in 100 cc bottles (Abbott)

As advocated by Griffith[37] and Walton[38] all these drugs and solvents are autoclaved in the trays together with the necessary syringes and needles Autoclaving in this fashion serves a dual purpose first it precludes infection should the pharmaceutical manufacturer dispense a contaminated solution and second it resterilizes the crystals should a small invisible crack in the glass occur during handling of the sealed ampule Autoclaving also sterilizes a gummed label while cold sterilization usually does not[4]

The following drugs may also be autoclaved at least once as described above (1) Pontocaine 1% solutions (2) Novocain 1 or 2% solutions (3) ephedrine sulfate solutions (4) Methedrine solutions (5) Vasoxyl solutions (6) Nupercaine solutions (7) Dola

min (ammonium sulfate) solutions (8) oil solutions (Proctocaine, etc), and (9) absolute alcohol[38, 39, 6] Note that ampules of Pontocaine, Novocain and Metycaine may be subjected to sterilization at least five times without altering the potency of the drug but local anesthetic solutions containing dextrose as well as the individual ampules of 10% dextrose may turn color (carmelize) if subjected to heat sterilization more than once[39] *Hyaluronidase cannot be heat sterilized at all*

Often Adrenalin may be mixed with the local anesthetic solution in order to prolong spinal anesthesia and it is usually emphasized that Adrenalin 1 1000 will decompose if subjected to heat sterilization and therefore must be cold sterilized Nevertheless Saklad[40] told the author that Adrenalin may be heat sterilized at least *once* without undergoing a chemical change and since then we have subjected this drug to autoclaving No alterations in the effectiveness of the Adrenalin in prolonging spinal or epidural anesthesia or peripheral nerve blocks have been noted Massa s [3] report confirms Saklad s and our findings concerning Adrenalin To obtain concrete evidence of this ampules of Adrenalin were autoclaved at the Mason Clinic by the above described method and sent to Burroughs Wellcome & Co for analysis to see if the potency was altered by a single autoclaving The answer read "I have just received the assay report of the two ampules of Parke Davis Adrenalin 1 1000 that you submitted to us Our assay revealed the following information

R 134 L 100 6 ± 2 0% of labeled potency
R 140 L 99 6 ± 2 8% of labeled potency

"From these data it would appear that solutions of epinephrine can be autoclaved at least once without any drop in potency As a matter of fact, Dr C W Ferry of our Control Laboratories has informed me that we used to autoclave our ampules of epinephrine Some of these appeared discolored after autoclaving but there is evidence that even those ampules which turned slightly yellow still retained adequate potency for clinical use"[54]

Tainter [-] also confirmed the fact that Adrenalin could be heat sterilized and added that this was true of Neosynephrine as well He emphasized that only when these two agents turn from a clear to a colored solution are they losing their full potency, and even then they maintain enough of their potency to give satisfactory clinical results

Although refinements in cold sterilization of drugs have been introduced by Nicholson and Eversole[41] Steinberg[4] and Lewis[43] *autoclaving drugs to be used in regional nerve* block is the accepted technique of sterilization at the Mason Clinic at the present time Until recently cold sterilization of the ampules of drugs employed at the Mason Clinic had been used No untoward complications from this occurred but the absence of complications was no reason in our estimation not to change to what logically is a better technique of sterilization and one which seems to be above reproach As the Management Methods Magazine for December 1954 commented "When something has been done in a particular way for 15 or 20 years it is a pretty good sign in these changing times that it can be done a better way"

Scrub Hands and Use Sterile Gloves—*Scrubbing of Hands*—Whether or not the physician should scrub his hands in the same manner as if he were going to perform a surgical procedure is a debatable question If he should, then the brushes to be used should have been sterilized and not previously used Vuylsteke[14] believes that the cause of two of his cases of meningitis could be traced to the use of a brush which the surgeon had previously used to clean his hands

Gloving—It is the author s opinion that sterile gloves should always be worn when a block is being performed First they are a definite aid in preventing dermatitis from local anesthetic drugs (see Chapter 14 Dermatitis page 127) and second they are an added factor of safety in avoiding infection They are no more a hindrance to the anesthesiologist performing a block than to the surgeon performing a delicate operation

Avoid Contaminating Blocking Solutions with Solutions Used to Prepare the Skin—

The medicine cup should be placed in front of the mixing graduate so that the prep solution will never pass over the local anesthetic solution If any alcoholic solution is dropped in the anesthetic solution and goes unnoticed a slough of the tissue a meningitis meningeal irritation or a neurological complication may ensue

Use Aseptic Technique when Opening Tray—Sterile forceps should be used to handle the equipment The antiseptic solution in which such forceps are often sterilized must not drip on the tray

Cleanse the Skin Prior to Needle Puncture—The skin should be painted with one of the readily available antiseptic solutions such as Merthiolate or Zephiran More important however is the fact that sufficient time should be allowed for the antiseptic solution to exert an effect

Touch Only Sterile Articles Once Gloved—Once the anesthesiologist has put on sterile gloves he must watch carefully that he touches only the sterile equipment

Use Introducer Prior to Insertion of Spinal Needle—At the Mason Clinic a Sise introducer is always used so that the tip of the spinal needle never touches the patient s skin If an introducer is not employed or if the stylet of the needle is not kept in place when the needle is inserted it is possible for the spinal needle to effect a punch biopsy of the skin as it is inserted Dickson[44] a British pathologist writes "In the centrifugalized deposits of lumbar puncture specimens I have not infrequently encountered squamous cells from the skin surface together with their accompanying staphylococci etc and occasionally even little cylindrical fragments of skin punched out by the exploring needle

Avoid Repeated Traumatic Punctures—Repeated efforts at spinal puncture may cause bleeding Repeated trauma offers more opportunity to contaminate the needle while blood furnishes an excellent growth media for bacteria

Do Not do a Spinal or Epidural Puncture if Patient s Bleeding Time is Increased—A blood dyscrasia or anticoagulant therapy predisposes to bleeding if a blood vessel should be punctured therefore spinal and epidural blocks are contraindicated for these patients Bonica[8] cites a case of paraplegia which resulted from subarachnoid hemorrhage following spinal anesthesia in a patient with a blood dyscrasia (page 89)

Avoid Spinal or Epidural Blocks in Patients with a Bacteremia—Not to do so is to invite a severe meningitis

Never Insert a Needle Through an Infected Area—If this is done the bacteria responsible for the infection may be easily carried into the epidural or subarachnoid space and result in a meningitis

Avoid the Use of New Local Anesthetic Agents—New local anesthetic drugs must not be used unless adequately and thoroughly tested Masson[4] states, "The search for new and better drugs goes constantly on but their experimental use should be restricted to institutions with proper facilities for their correct evaluation Only for sound reasons should a well established drug be abandoned for an unproven one

TREATMENT

Before instituting extensive treatment when the signs and symptoms of a meningitis or a simple meningeal irritation occur a spinal puncture should be made to establish a definite diagnosis

Aseptic Meningitis or Meningeal Irritation—If an aseptic meningitis or only meningeal irritation is discovered with no signs of a generalized toxicity, symptomatic therapy good nursing care and watchful waiting may be sufficient These cases usually improve within 2 to 4 days after onset of the signs and symptoms The one case of subarachnoid hemorrhage which has occurred at the Mason Clinic was satisfactorily treated by careful observation and waiting However if the patient shows a systemic toxicity i e fever etc and the cell count of the cerebrospinal fluid is increased the physician although the smear of the spinal fluid is negative probably should not wait for the spinal fluid to be cul

tured but should place the patient on antibiotics immediately and treat him just as if septic meningitis were present

Septic Meningitis.—Once this diagnosis is established intensive therapy must be instituted It consists of

General Measures.—Good nursing care is essential The patient should be confined in a dark quiet room Sedatives and opiates may be necessary to control headache restlessness and occasionally convulsions Bladder distention and constipation must be guarded against Nutrition and fluid intake must be maintained by tube feeding and intravenous therapy if necessary

Specific Measures.—If the bacteria involved can be isolated, the appropriate antibiotic should be used If they cannot be isolated if the signs and symptoms become progressively more severe (precluding an aseptic meningitis) and if the physician is not willing to wait for the cultures of spinal fluid to reveal the specific organisms involved then the antibiotics with the widest spectrums should be used Penicillin certain sulfa drugs (diazine) streptomycin Terramycin chloramphenicol and polymyxin B either alone or in combination have been the most useful drugs since they enter the spinal fluid in high concentrations [46] Systemically administered Terramycin was originally thought not to enter the cerebrospinal fluid readily [47] Therefore it was not advocated as a satisfactory drug to use in septic meningitis but recently the efficacy of Terramycin therapy in treating this disease has been noted [48] Lavage of the subarachnoid space by placement of these drugs into this space is no longer considered necessary

The prognosis of meningitis following a regional block procedure must be guarded but with improved antibiotic therapy the death rate and incidence of sequelae have decreased The most frequent sequelae are deafness blindness aphasia, peripheral neuritis paralysis endocarditis and arthritis [20 21]

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tured, but should place the patient on antibiotics immediately and treat him just as if septic meningitis were present

Septic Meningitis —Once this diagnosis is established intensive therapy must be instituted It consists of

General Measures—Good nursing care is essential The patient should be confined in a dark, quiet room Sedatives and opiates may be necessary to control headache restlessness and occasionally convulsions Bladder distention and constipation must be guarded against Nutrition and fluid intake must be maintained by tube feeding and intravenous therapy if necessary

Specific Measures—If the bacteria involved can be isolated the appropriate antibiotic should be used If they cannot be isolated if the signs and symptoms become progressively more severe (precluding an aseptic meningitis) and if the physician is not willing to wait for the cultures of spinal fluid to reveal the specific organisms involved then the antibiotics with the widest spectrums should be used Penicillin certain sulfa drugs (diazine) streptomycin Terramycin chloramphenicol and polymyxin B either alone or in combination have been the most useful drugs since they enter the spinal fluid in high concentrations[46] Systemically administered Terramycin was originally thought not to enter the cerebrospinal fluid readily[47] Therefore it was not advocated as a satisfactory drug to use in septic meningitis, but recently the efficacy of Terramycin therapy in treating this disease has been noted[48] Lavage of the subarachnoid space by placement of these drugs into this space is no longer considered necessary

The prognosis of meningitis following a regional block procedure must be guarded but with improved antibiotic therapy the death rate and incidence of sequelae have decreased The most frequent sequelae are deafness blindness, aphasia peripheral neuritis paralysis endocarditis and arthritis[9, 21]

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proach (3) the physical status of the patient and (4) the operative risk.* The conclusion of Dripps and Vandam's [9] article in which 10,098 cases of spinal anesthesia are reviewed is of interest "Anesthesia of any type places a stress on the patient A price is exacted for the pain relief and improved surgical working conditions that are provided during operation Continuing efforts must be made to compare anesthetic agents and techniques so that this price can be assessed Our experience indicates that the mortality rate following spinal anesthesia is lower than that recorded after general anesthesia in comparable patients undergoing comparable types of operations Similar observations are made in the current analysis of deaths associated with anesthesia reported by Todd and Beecher The most vociferous objection to spinal anesthesia comes from those who believe that chronic incapacitating neurological disease occurs with sufficient frequency after spinal anesthesia to make its use unjustified except in rare instances It is not scientific thinking always to attribute to the anesthetic a neurologic complaint arising in a patient who has had spinal anesthesia Other diagnostic possibilities must be kept in mind constantly lest specific therapy be withheld because of an error in diagnosis Physicians appear prone to assume a cause and effect relationship between spinal anesthesia and a variety of complaints sometimes appearing years after the anesthesia This attitude is not objective nor is it justified on the basis of available data Some have taken the position that spinal anesthesia will soon be of historic interest only The results of this long term study do not support such a position they even suggest the possibility that such an attitude might deprive the patient of a safer method of anesthesia

This chapter is not slanted to support the advocates of spinal anesthesia or to try to "bury the technique for its adversaries However it behooves the physician employing spinal block epidural block or paravertebral block to acquaint himself with the causes as well as the possible prophylaxis of the most serious of all neurological complications which may follow them—spinal cord paralysis and/or death The other neurological complications from these regional blocks i e headache, dizziness cranial nerve paralysis or paresis meningitis (septic meningitis excluded) and neuritis usually leave no permanent sequelae Unfortunately this is not true of involvement of the arachnoid or the parenchyma of the spinal cord which instead of improving may progress in severity

ETIOLOGY

From clinical observations as well as autopsy reports the pathological change which occurs in the greater number of the severe spinal cord complications, i e, cauda equina syndrome paraplegia quadriplegia and death is an adhesive proliferating arachnoiditis and/or change in the spinal cord itself [1, 2, 8, 27] These pathological changes are usually the most marked at the level where the anesthetic solution has been injected and consist mainly of (1) softening of the cord [15, 18] (2) myelin sheath, axis cylinder and glia destruction at the periphery of the cord and at the entrance of the posterior roots [18, 19] (3) hyalinization of the pia and arachnoid, [18] (4) damage to both the white and gray matter [18] (5) hyaline changes in blood vessels, [8] and (6) ascending myelitis [5, 20] Williams [2] believes that when the picture of chronic arachnoiditis develops, these devastating effects upon the neural structures are most probably due to decreased blood supply and starving of the nervous tissue

The causes of these pathological changes are open to debate but it appears that one or more of the following may be *responsible* (1) the destructive effects of local anesthetic agents on the *leptomeninges and spinal* cord (2) improper sterilization of drugs or equipment (3) undiagnosed neurological diseases (4) trauma (5) bacterial contamination of the subarachnoid *space* (6) *subarachnoid* in

* Risk not only includes the ability of the anesthetist and the patient's physical status but also the hospital facilities available ability of the surgeon etc

CHAPTER 23

Lesions of the Spinal Cord Following Regional Block Procedures

When anesthetic agents are injected purposely into the subarachnoid space or reach it and the parenchyma of the spinal cord following local infiltration peripheral nerve block or epidural block injury to the spinal cord may ensue This injury may range from paralysis of a single nerve to a cauda equina syndrome or an ascending arachnoiditis with paraplegia quadriplegia or death[1 ?] The serious nature of these complications has led Kennedy *et al*[1] to write From a neurological point of view we give the opinion that spinal anesthesia should be rigidly reserved for those patients unable to accept a local or general anesthetic Paralysis below the waist is too large *a price for a patient to pay in order that* the surgeon should have a fine relaxed field It is neither the purpose nor the intent of this chapter to support or take issue with such a statement The medical literature cited above is ample evidence of the occurrence of these very serious neurological complications

Spinal anesthesia is being extensively used in many hospitals throughout the country and it does not appear that this situation will change markedly in the near future Whether this is a fortunate or unfortunate situation is still a hotly debated question In all fairness to spinal anesthesia it must be said that paralysis following spinal anesthesia is relatively infrequent when the thousands of cases performed each year without incident are considered It must also be noted that cases of serious neurological complications following general anesthesia have been reported[2 26 47 111 11] In most instances the pathological changes seen after inhalation anesthesia are a result of brain damage and not a result of destructive changes in the spinal cord which are usually the type that spinal anesthesia may bring about

Paralysis of the brachial plexus its branches or of other nerves which are easily involved by pressure alone should not be automatically thought of as a complication of the anesthetic procedure per se when it follows a regional block procedure and surgery (see Chapter 11 page 121) Such paralysis is usually caused by position and may follow both general and regional anesthesia It should also be noted that deaths from cardiac failure explosions respiratory depression vomiting with aspiration and unknown reasons (so called "anesthetic deaths) occur with as much or greater frequency after general anesthesia as paralysis following spinal[48 49] However the unfortunate consequences of general anesthesia are literally buried and forgotten while with the regional block procedures the patient often lives to haunt the physician and make him more acutely aware of the unfortunate occurrence

Since no anesthetic method is 100% safe and since neurological disorders may follow either general or regional anesthetic procedures the arguments as to which is the safer anesthetic method would seem to resolve itself to the following considerations (1) who is administering the anesthetic (2) whether or not his technique is above re

or symptoms On the other hand, in the exceptional case, for some unknown reason the patient's physiological response is abnormal—and this inflammatory reaction does not subside but progresses When this relatively rare situation develops either a chronic arachnoiditis (leptomeningeal involvement) results with a slow onset of spinal cord pathological changes or an immediate neurolytic effect on the parenchyma of the cord per se ensues with a rapid onset of paralysis

Improper Sterilization of Drugs and Equipment—If the ampules of the drugs used for spinal anesthesia are soaked in neurolytic drugs e g alcohol phenol or formaldehyde and there should be an invisible crack in the ampule these drugs may seep into the ampule If this is not noticed at the time the drug is mixed the adulterated drug may damage the cord [6, 64]

Moreover several special blood solvents and detergents have been devised to cleanse instruments i e Haemo Sol and Detergex etc If these solutions are not adequately rinsed from the needles and syringes their neurolytic action on the cord may result in severe neurological complications of the central nervous system [12] In the same way if syringes and needles are cold sterilized and inadequately rinsed prior to use in regional block, neurological damage may result from the sterilizing solution

Heat sterilization will avoid both of these!

Undiagnosed Pre-existing Neurological Diseases—A pre existing neurological disease such as pernicious anemia multiple sclerosis tabes general paresis or cord tumors may be dormant until a lumbar tap or a spinal anesthesia has been executed In other words the spinal anesthetic acts as the precipitating factor [3, 7, 20, 21, 65, 66] The author has seen two such cases one patient had multiple sclerosis and the other amyotrophic lateral sclerosis

The appearance of signs and symptoms of spinal cord tumors after tap of the dura is presumably due to the changes in the spinal fluid dynamics which the tap produces These changes result in vascular engorgement and edema of the unsuspected tumor and/or its displacement downward, creating a block One or both of these then brings out the previously latent signs and symptoms of a pre existing tumor [67]

Trauma to the Cord or Nerve Root—During a spinal or epidural tap either a nerve root or the cord itself may be impaled by the point of the needle The more cephalad the tap is performed above the third lumbar interspace the greater are the chances of traumatizing the cord According to Reiman and Anson's [68] examination of 129 adult cadavers the spinal cord ends at or above the level of the body of the second lumbar vertebra in 94% of humans In a few, it extends caudally to the level of the middle of the body of the third lumbar vertebra

When the needlepoint touches or impales the nervous tissue, the patient usually complains of paresthesias in the areas innervated by the nerve or nerves In the lumbar region these usually are represented by "electric shock like" sensations in the bladder, rectum genitalia or lower extremity If the needle is withdrawn at this time, permanent sequelae usually do not result On numerous occasions while placing a spinal needle the author has elicited paresthesias of the nerves of the cauda equina and has readjusted the needle To date none of these cases has resulted in nerve involvement Orkin *et al* [69] cite a case in which the needle was inadvertently inserted into the spinal cord during an attempt to execute a left stellate ganglion block by the posterior thoracic approach The patient complained of severe pain as the needle inadvertently pierced the cord and the procedure was discontinued no anesthetic solution having been injected Anesthesia was noted on the left side down to the umbilicus that day The next day no residual anesthesia was present On the other hand, neurological sequelae, though rare, may result Maxson [70] cites cases of impaling of the cord or its nerve roots with residual sciatic pain Likewise Bonica [71] relates a case in which an epidural block was being attempted in the thoracic region and the spinal cord was inadvertently impaled while placing the needle The patient com

jection of vasopressor drugs, (7) subarachnoid injection of neurolytic drugs and (8) unusual bleeding tendencies

The Destructive Effect of Local Anesthetic Agents on the Leptomeninges and Spinal Cord—Merritt and Fremont Smith[1] have noticed that when any sterile foreign substance —this would include a local anesthetic agent —is injected into the subarachnoid space an aseptic meningeal reaction results It has been shown experimentally in animals that when local anesthetic agents are injected in high concentrations subarachnoidally they may cause leptomeningeal inflammation and damage to the parenchyma of the spinal cord[8, 9] Kamsler[60] notes that inflammation of the leptomeninges is a constant concomitant of spinal anesthesia in man

Davis *et al*[7] showed that when Nupercaine Spinocain, Gravocaine or Novocain (procaine) were injected in *high* concentrations subarachnoidally in dogs the following changes occurred (1) the leptomeninges showed varying inflammatory changes (2) the ganglion cells of the gray matter showed wallerian degeneration (3) the axis cylinders became swollen and fragmentation occurred and (4) myelin degenerative changes in the fiber tracts of the spinal cord ensued Friedman[61] *has confirmed the fact that local anes*thetic agents do have a myelolytic effect on the cord and points out that the peripheral and posterior structures of the cord are the most susceptible During regional block analgesia the only conditions under which an unusually excessive volume of local anesthetic drug might reach the subarachnoid space and/or parenchyma of the spinal cord is when it is inadvertently injected subarachnoidally while performing an epidural (peridural) or paravertebral block or if an overdosage is by error placed in the subarachnoid space

The reader should remember that drugs injected paravertebrally may reach the spinal cord via (1) long dural cuffs which extend past the intervertebral foramina (2) misplaced needles which have been directed too medially and have passed between the neural arches or through the intervertebral foramina and punctured the dura or (3) the perineural spaces (see Chapter 5 page 48) However even when a mistake of this nature is made, spinal cord damage does not usually follow

The reported cases of paralysis following spinal anesthesia in man would indicate that the anesthetic agent acting as a chemical irritant is to blame for most of the pathological changes Autopsy reports and investigative laminectomies have revealed that the greatest degree of damage occurs in the lumbar area in the vicinity of the spinal tap This would be the area subjected to the highest chemical concentration of the local anesthetic agent In the literature reviewed the one case of severe damage to the spinal cord other than in the lumbar area followed an inadvertent subarachnoid injection of local anesthetic solution while a paravertebral block of the upper intercostal nerves was being performed In this situation a residual paralysis resulted which was confined to the nerve roots of both brachial plexuses This confined damage would confirm the statement that the drug per se is to be blamed[11] When spinal cord involvement does occur either the local anesthetic drug or other drugs (lipiodol propylene and polyethylene glycol the sulfonamides *penicillin or streptomycin*) which may act as chemical irritants have been injected sub arachnoidally[3, 6, 63] Spinal cord involvement rarely follows diagnostic spinal tap unless preexisting undiagnosed pathology of the spinal canal or its contents is present

If the local anesthetic agent is the causative factor the question then arises why are thousands of spinal anesthesias performed per year and the number of reported cases of spinal cord damage so small? Allergy to the drug itself has been ruled out[2] It is the author s opinion that this again may be a case of "hyperergy" (see Chapter 1 page 8 for the definition of hyperergy) In other words the safe spinal dosage of the local anesthetic agent normally causes only a very mild inflammatory reaction This usually results in a small increase in the cells (50 to 150) in the spinal fluid alone and produces no other signs

the sacral arteries. Cases have appeared in the literature of transverse myelitis following the clamping of a spinal branch of these arteries during sympathectomy and were attributed to the fact that the anterior spinal artery was not a continuous artery as usually described but was anomalous.[84] Haven,[84] Ward[8] and Foltz[86] point out that if a vasoconstrictor drug were used subarachnoidally in a patient with such an anomalous blood supply to the spinal cord and prolonged vasoconstriction of the arteries to a section of the cord ensued, results duplicating those seen following sympathectomy could occur.

In monkeys, Wu *et al*[63] were able to show neurological changes from the continuous subarachnoid injection of Adrenalin and Neosynephrine. Nevertheless this occurred only after doses ten to several hundred times the clinical dose were used continuously over long periods of time. From this research and our clinical experiences at the Mason Clinic as well as those of others cited previously, it seems reasonable to conclude that when these two time-tested vasoconstrictor agents are used subarachnoidally to prolong *single* dose spinal anesthesia the chances are infinitesimal that these drugs will produce spinal cord damage.

On the other hand the newly introduced vasoconstrictor drugs, i.e., Vasoxyl, Levophed and Wyamine, agents with a prolonged vasoconstrictor action, should not be used subarachnoidally to prolong spinal anesthesia until further experimental research assures their safety when used in this fashion. The neurologist of the Mason Clinic has seen one case of cauda equina syndrome (at another hospital) which followed the subarachnoid administration of one of these drugs. In this case 0.5 cc. of Vasoxyl had been mixed with the local spinal anesthetic solution in hopes of prolonging anesthesia time for an appendectomy.[84] This does not necessarily condemn the Vasoxyl since the paralysis may have been caused by other factors; nevertheless, the action of this drug must be considered as a possible factor.

Injection of Neurolytic Drugs into the Subarachnoid Space.—When neurolytic drugs gain entrance to the subarachnoid space or the parenchyma of the spinal cord, the reason for the sequela is obvious. Further comment is superfluous other than to point out again that (1) drugs injected paravertebrally may reach the spinal cord via (a) dural cuffs which extend past the intervertebral foramina, (b) misplaced needles which have inadvertently punctured the dura, or (c) the perineural spaces (see Chapter 5, page 48) and that (2) alcohol, phenol, oil preparations of various kinds (e.g., Bromsalizol), propylene glycol and polyethylene glycol are neurolytic drugs.[90–118]

Increased Bleeding Tendencies.—If a patient has a blood dyscrasia or has been receiving anticoagulant therapy and an epidural or spinal blood vessel is traumatized, bleeding into the epidural or subarachnoid space may result in neurological complications. Bonica[89] cites such a case and states, "The author encountered one such case in which a block was administered to a surgical patient who had prolonged bleeding time unknown to the surgical team. Four days following a bloody spinal tap the patient began to have signs of the cauda equina syndrome which was found to be due to pressure from large clots in the subarachnoid space."

SIGNS AND SYMPTOMS

The signs and symptoms of neurological complications involving the parenchyma of the spinal cord and the nerve roots depend on whether the neural tissue itself or its coverings (the leptomeninges) are primarily involved.

Damage to the Nerve Root or Parenchyma of the Spinal Cord Only.—*Onset and Extent of Paralysis.*—In these cases the onset is immediate and the paralysis is apparent from the moment the spinal anesthetic has dissipated itself. Note again that if individual nerve paralysis occurs following spinal analgesia, for example, paralysis of one of the nerves of the brachial plexus, the lateral femoral cu

plained of severe electric shock like pain from the level of the tap down. No solution was injected and a general anesthetic was administered. At the writing of this chapter, seven months later the patient has pain and an area of numbness of the dermatomes on the left side of the abdomen. Motor and sensory function of the bowel, bladder, lower extremities, etc. are normal.

It should be stressed that if the needle is not readjusted or the procedure discontinued when a spinal nerve root or the cord is impaled serious sequelae may develop from injecting the anesthetic solution. This may be due either to mechanical pressure or to the drugs used. If only a nerve root is involved the patient will complain bitterly of pain as the solution is injected and will in all probability show a residual impairment of sensations of the lower extremities, bladder or rectal paralysis, foot drop and/or trophic disturbances. Most of the articles cited herein report this type of complication. If on the other hand an injection into the spinal cord parenchyma is made, the patient may suddenly collapse into unconsciousness and if he does not die a transverse myelitis will usually develop.[7] Thorsen[illegible] cites seven such cases.

Other than these specifically noted types of trauma it is unlikely that multiple punctures of the dura or tearing of a subarachnoid blood vessel can cause severe permanent spinal cord injuries unless other diseases are present, e.g. increased bleeding time. Only one possible case of medullary compression from subdural bleeding in a presumably normal adult was found in the literature.[113] Even protrusion of an intervertebral disc following trauma by the spinal needle is not a permanently disabling complication.

Bacterial Contamination of the Subarachnoid Space.—The sequelae of a septic meningitis while infrequent, are well known. They consist mainly of a chronic arachnoiditis, external hydrocephalus and root involvement and these in turn may lead to paralysis, blindness, cauda equina syndrome, etc.

Subarachnoid Injection of Vasoconstrictor Drugs.—Vasoconstrictor drugs have been injected subarachnoidally after being mixed with the local anesthetic solution in an effort to prolong the duration of anesthesia. Adrenalin, Neosynephrine and ephedrine sulfate have been quite effective in accomplishing this aim and to date no untoward spinal cord damage from their use has been reported.[73, 83, 114] Nevertheless, the neurologists who work at our hospital are sceptical about the use of a vasoconstrictor drug subarachnoidally.[81, 86] They base their reasoning on abnormalities in the anatomy of the blood supply to the spinal cord.

The normal blood supply to the spinal cord is described by Gray[87] as follows: "The Anterior Spinal Artery (a. spinalis anterior; ventral spinal artery) is a small branch which arises near the termination of the vertebral and descending in front of the medulla oblongata, unites with its fellow of the opposite side at the level of the foramen magnum. One of these vessels is usually larger than the other but occasionally they are about equal in size. The single trunk thus formed descends on the front of the medullar spinalis and it is reinforced by a succession of small branches which enter the vertebral canal through the intervertebral foramina; these branches are derived from the vertebral and the ascending cervical of the inferior thyroid in the neck, from the intercostals in the thorax and from the lumbar iliolumbar and lateral sacral arteries in the abdomen and pelvis. They unite by means of ascending and descending branches to form a single anterior median artery which extends as far as the lower part of the medulla spinalis and is continued as a slender twig on the filum terminale. This vessel is placed in the pia mater along the anterior median fissure; it supplies that membrane and the substance of the medulla spinalis and sends off branches at its lower part to be distributed to the cauda equina."

While in most patients this artery is continuous anomalies do occur and in these instances the anterior spinal artery is not always a continuous structure. When it is not continuous sections of the cord may derive their arterial supply directly from the spinal branch of the intercostal arteries, lumbar arteries, and

the sacral arteries Cases have appeared in the literature of transverse myelitis following the clamping of a spinal branch of these arteries during sympathectomy and were attributed to the fact that the anterior spinal artery was not a continuous artery as usually described but was anomalous[88] Haven[84] Ward[8] and Foltz[86] point out that if a vasoconstrictor drug were used subarachnoidally in a patient with such an anomalous blood supply to the spinal cord and prolonged vasoconstriction of the arteries to a section of the cord ensued results duplicating those seen following sympathectomy could occur

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taneous nerve or the peroneal nerve, it is by all odds a positional paresis and the anesthesia should not be immediately condemned.

The paralysis when due to a spinal block procedure may involve only a single nerve or a group of nerve roots, i.e., only a foot drop may occur. Cases of incontinence, rectal paresis or sexual impotency alone following spinal anesthesia have also been reported.[97, 98] On the other hand, a cauda equina syndrome or paraplegia may develop. A cauda equina syndrome consists of the following: (1) retention of urine, (2) incontinence of feces, (3) impairment of sensations in the perineal area and buttocks, (4) impotency, and (5) motor weakness and altered reflexes of the lower extremities.

Course.—If only a single nerve or a few nerve roots are involved, as when a foot drop results, complete recovery may occur in six months to a year. Ferguson and Watkins,[8] Nicholson and Eversole[3] and Brown[33] report cases of foot drop following spinal anesthesia in which the patients recovered in this period of time. Palmer, writing in Pitkin,[9] reports three cases of urinary incontinence alone as a result of loss of sphincter control, and deCarle[99] reports one such case. All cases recovered in a "few months." Loss of potency has been attributed to spinal anesthesia, but this is questionable, for considerable psychotherapy alone may correct the problem.[97] When only a cauda equina syndrome develops, some improvement of its symptoms, particularly those of bladder and rectal dysfunction, may ensue. But a case of paraplegia seldom improves markedly, although it does not usually become progressively worse as as does the granulomatous leptomeningeal proliferation of chronic arachnoiditis.

Spinal Fluid Findings.—Few changes are found in the spinal fluid, which is not difficult to obtain. The spinal fluid of a typical case 20 hours after anesthesia showed: (1) initial pressure of 50 mm. of water, (2) total protein 347 mg., (3) 21 mg. of sugar per 100 cc. of spinal fluid, and (4) lymphocytes.[3]

Surgical Explorations.—The lesions in these cases are most marked at the site of injection of the drug and limited to the nervous tissue of the cord and its nerve roots. The coverings of the cord, i.e., the dura and the arachnoid, are not markedly involved in the pathological changes.

Damage to the Leptomeninges.—*Onset and Extent of Paralysis.*—Following the spinal anesthesia there is usually a period during which the patient is symptom free. He has no indication of neurological involvement. Usually three or four days to a few months and in the occasional case one to two years after the spinal anesthetic procedure was executed, he begins to develop pain, sensory loss or weakness of one or both legs.[22] The bladder and rectum may or may not be involved at the onset.

Williams[27] states: "The symptoms [in chronic arachnoiditis] result from the constrictive effect of the arachnoiditis upon the vascular channels of the spinal cord, the least competent of which are in the mid dorsal region." He believes that this explains why the neurological findings in these cases often suggest a tumor at the level of the sixth thoracic vertebra. The clinical picture in these cases is that of a transverse myelitic lesion, not that of the lower motor neuron involvement which accompanies the cauda equina syndrome.

Course.—One of the characteristics of the arachnoiditis is that it is chronic and progressively becomes more severe. The arachnoiditis may become stationary after a certain time and then regress slightly, but it may also progress to obliterate the entire subarachnoid space and may eventually cause death.[1, 27] Seldom, once the paralysis has occurred, is recovery ever remarkable.

Williams, in referring to this slow onset, states: "All 9 patients recovered from their initial operations and anesthesia and left the hospital without neurologic symptoms. This is probably the most sinister aspect of this disorder, for those who performed the operations and gave the anesthetics are probably unaware that their services carried later reprisals."

Spinal Fluid Findings.—In most of these cases it is *impossible* to obtain spinal fluid because of the chronic proliferative arachnoid

this However if a tap is successfully executed evidence of a block of the spinal fluid is present

Surgical Exploration —When viewed grossly following a laminectomy a dense arachnoiditis is seen Microscopically there is seen a dense collagenous fibrous cuff surrounding and constricting the spinal cord and adjacent vessels and containing in its interstices scattered aggregations of inflammatory cells

PROPHYLAXIS

Of course the best prophylaxis against any regional block complication is not to perform the block Nevertheless this is often an impractical attitude Moreover the author knows of no procedure in medicine that is 100% safe 100% of the time And since spinal anesthesia appears to be gaining popularity among physicians at the present time rather than losing it it behooves the physicians using this type of analgesia to see that the following prophylactic steps are rigidly observed Most of these prophylactic measures have previously been enumerated by Nicholson and Eversole[3] Griffith,[100] Anderson[101] and Macintosh[10]

Carefully Clean and Sterilize all Needles and Syringes—Avoid the use of detergent compounds when washing instruments and use heat sterilization methods (see Chapter 22 page 206)

Avoid Contamination of the Drugs Either by Bacteria or Neurolytic Agents—Autoclaving drugs to be injected in the subarachnoid space is today the preferred method of assuring sterility and preventing the neurolytic agents often used in cold sterilization techniques from being inadvertently placed subarachnoidally (see Chapter 22 page 207)

Supposedly improved methods of cold sterilization including deep coloration of the sterilizing solutions have been advocated as simple expedients to reduce the unnoticed contamination of these drugs by the sterilizing solution[8,103,104,10] However these techniques are not as fool proof as heat sterilization and may give the physician a false sense of security[100,105,106,107]

Identify all Drugs Prior to Subarachnoid Use—Hospital prepared solutions and solutions not clearly labelled should not be accepted for injection

Discard Turbid or Cloudy Solutions—Solutions which have become turbid or contain undissolved crystals should not be employed

Do Not Perform Spinal Puncture in the Presence of Infection—If the patient has a bacteremia or the area to be entered by the needle is infected use another type of anesthesia

Avoid Spinal Anesthesia in Presence of Known Spinal Cord Disease, Virus Infections, and Intractable Back Pain—Tabes multiple sclerosis pernicious anemia spinal cord tumors metastatic vertebral lesion acute poliomyelitis and chickenpox are contraindications to spinal anesthesia[3,21] Michelsen[21] cites four cases of intraspinal tumors which were diagnosed only after a spinal anesthesia accentuated the symptoms He felt that the diagnosis could have been made by the physician performing the spinal tap in two of the cases prior to the administration of the local anesthetic drug In one of these two cases the spinal fluid was discolored and in the other it was under marked pressure

Do Not Inject Local Anesthetic Solution Unless a Free Flowing Clear Spinal Fluid Is Obtained—Persistently bloody tap or xanthochromic spinal fluid is a definite contraindication to injecting the local anesthetic solution Or if paresthesias are elicited during the tap and/or a free flow of spinal fluid is not obtained the needle should be readjusted to overcome these problems If this cannot be accomplished another type of anesthesia should be used

If severe paresthesias result as the drug is being injected the injection should be halted and the needle readjusted Then if paresthesias still occur, the procedure should be discontinued Maxson[70] stresses the use of a short beveled spinal needle to reduce the incidence of impaling the spinal cord and its nerve roots

Use the Smallest Applicable Dosage of a Time Tested Local Anesthetic Agent which Will Produce the Desired Effect—Since local

anesthetic agents are neurolytic in high concentrations and since some patients may exhibit the phenomenon of hyperergy (see page 8) the lowest possible dosage and concentration of a local anesthetic drug which will give the desired anesthesia should be employed The maximum concentrations of drugs commonly used in spinal anesthesia are (1) 0 5% Pontocaine (2) 5% Novocain (3) 4% Metycaine and (4) 0 1% Nupercaine[101]

Do not use every new local anesthetic drug made available commercially for spinal or epidural analgesia The time tested drugs such as Pontocaine Novocain etc should not be discarded too readily for new drugs unless these new drugs have been tested extensively experimentally and have proven their definite advantage over the drugs that have stood the test of time (see Maxson s statement page 209) To date my associates and I have seen three cases of prolonged numbness attributable to the local anesthetic agent following epidural (peridural) block and none following spinal (subarachnoid) block All three occurred following the use of 2% Cyclaine for caudal block One of these persisted for one week and involved the area around the anus The other two resolved within 48 and 72 hours respectively One of these two involved the area innervated by the right lateral femoral cutaneous nerve and the other involved the skin of the buttocks

Do Not Inject the New Powerful Vasoconstrictor Drugs with a Prolonged Duration Subarachnoidally —Vasoxyl Levophed or Wyamine should not at the present time be substituted for Adrenalin Neosynephrine or ephedrine sulfate to prolong subarachnoid anesthesia Their powerful vasoconstrictor action may produce a prolonged constriction of the anterior spinal artery resulting in hypoxia or anoxia of the cord

Lavage the Subarachnoid Space if a Spinal Fluid Block Is Suspected —If the desired level of analgesia is unobtainable by the ordinary methods one should consider the possibility of a spinal fluid block To remove any residual anesthetic solution in these cases, Nicholson and Eversole[3] advise flushing out the subarachnoid space with isotonic saline

If There Exists a Previous History of Difficulties with a Spinal Anesthetic, Avoid Spinal Analgesia —If the history of the patient reveals delayed return of motor or sensory function following a previous spinal, choose some other type of analgesia

Do Not Force a Patient to Have a Spinal Anesthetic —This type of patient is a constant complainer and if any residua from a spinal block develop legal action is unavoidable Griffith[100] states " there are some patients both in obstetrics and in surgery, who simply do not want a spinal anesthetic under any circumstances For these patients, unless there is some vital reason for choosing spinal anesthesia it is foolish for the anesthetist to insist Let us sensibly choose some other method A nervous patient who has been persuaded against his will to undergo spinal anesthesia will have a tendency to blame all his aches and pain for years afterwards to this procedure whether or not there is any logical connection It is equally foolish for an anesthetist to persist in unsuccessful attempts at lumbar puncture when a patient is becoming disgruntled and tense When spinal anesthesia is given to a patient against his will it may be interpreted by the court as technical assault"[103]

TREATMENT

When these unfortunate sequelae arise following a spinal anesthesia careful handling of the patient is essential A diagnostic lumbar tap exploratory surgery of the spinal cord or watchful waiting may be indicated Surgery seldom improves the patient and in many instances has made the condition worse This according to Williams[2] is singularly true in cases of chronic arachnoiditis However Ward[8] feels that he has improved some of these patients by freeing adhesions between the meninges and the spinal cord or its nerve roots

Certainly good nursing adequate care of the bladder and rectum a suitable psycho

logical approach to the situation and rehabilitation are all essential Therefore when a severe neurological complication occurs the patient should be referred to the neurosurgeon who is skilled in handling these problems The unfortunate physician who administered the anesthetic should also inform his lawyer or insurance company of the occurrence

COMMENT

Law Suits —In most instances when these complications occur and a law suit is instituted in the United States the patient is awarded the verdict on the basis of "Res Ipsa Loquitur" i e the condition speaks for itself, not on the fact that the physician has been negligent This is unfortunate because it encourages the patient to sue It is interesting to note that in England the court is prone to support the hospital and the physician when negligence cannot be proven[109, 110] The court decisions in two cases of paralysis from the waist down from Nupercaine contaminated with phenol solution which occurred in England indicated this and served to warn us of the danger of cold sterilization of ampules of drugs to be used in spinal anesthesia The following is quoted from the British Medical Journal[110]

"Lord Justice Denning said that no one could be unmoved by the disaster of these two men The fact called for an explanation by the hospital authorities and Dr Graham and they had given one They spared no trouble or expense to seek out the cause of the disaster There was a danger that the ampoules might become cracked and the cracks might be so fine that they could not be detected by ordinary inspection The carbolic disinfectant in which the ampoules were kept would then seep through the cracks into the nupercaine and no one would realize that it had taken place That was the explanation of the disaster and the question was whether any of the staff was negligent Medical science had conferred great benefits on mankind, but those benefits were attended by considerable risks Every surgical operation was attended by risks Dr Graham sought to escape the danger of infection by disinfecting the ampoule In escaping that known danger he unfortunately ran into another danger He did not know that there could be undetectable cracks but it was not negligent for him not to know it at that time We must not look at this 1947 accident with 1954 spectacles said his lordship The judge acquitted Dr Graham of negligence and we should uphold his decision Never again it was to be hoped would such a thing happen It was the extraordinary accident to these two men which first disclosed the danger Nowadays it would be negligence not to realize the danger but it was not then These two men had suffered such terrible consequences that there was a natural feeling that they should be compensated but we should be doing a disservice to the community if we were to impose liability on hospitals and doctors for everything that happened to go wrong Doctors would be led to think more of their own safety he continued than of the good of their patients Initiative would be stifled and confidence shaken A proper sense of proportion requires us to have regard to the conditions in which hospitals and doctors have to work We must insist on due care for the patient at every point but we must not condemn as negligence that which is only a misadventure"

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TABLE XXX

INCIDENCE OF ABDUCENS NERVE PALSY FOLLOWING LUMBAR PUNCTURE

[illegible]	[illegible]	[illegible]	[illegible]
Nicholson & Ivers	1,000	0	0
Avery et al[?]	[illegible]		0
Babcock[?]	2,000	[illegible]	[illegible]
Brown[8]	600		0.33
Sandberg & Bishop[?]	[illegible]	1	[illegible]
Faircloth	[illegible]	10	[illegible]
Kennedy & Lockhart[20]	[illegible]	2	0.13
Arner[4]	[illegible]	0	0
Greene et al[21]	[illegible]	0	0

by itself bring about the complication it is not unreasonable to expect that a combination of them may be responsible. The work of Arner[4] and Kennedy and Lockhart[20] substantiates this belief. Arner[4] states "... post puncture spinal fluid hypotension cannot be the only factor of significance in the genesis of palsy of the abducens and other cranial nerves since such pareses seem to be considerably more common after spinal anesthesia than after diagnostic lumbar puncture alone."

Since the abducens nerve is the cranial nerve most frequently affected, it is the most discussed, and while the factors listed below have been suggested in articles dealing with that specific nerve, they generally apply to most if not all cranial nerve paralyses or pareses.

Hypotension of the Spinal Fluid (Leakage Theory).—Most authors reporting cases of sixth nerve (abducens) injury seem to agree that the leakage of spinal fluid and the resultant low spinal fluid pressure is probably the most important single factor in producing this complication. The following statement of Greene *et al*[21] is typical of this belief: "Post puncture palsies of cranial nerves are primarily caused by the effect of gravity on a brain that has lost its intracranial water cushion by leakage of cerebrospinal fluid through a lumbar dural puncture in a patient whose state of hydration is inadequate to replace spinal fluid as rapidly as it is lost. In most patients the low intracranial volume of cerebrospinal fluid leads only to a postpuncture headache. In some, however, a cranial nerve palsy follows because of an anatomical or functional susceptibility of one or more cranial nerves, usually the abducens, to traction or pressure or both by the brain that is caudally displaced by severe or prolonged reduction of cerebrospinal fluid volume."

The theory that cranial nerve palsy is caused primarily by hypotension of the spinal fluid is based on the following observations:

Headache Precedes Nerve Paralysis.—In almost all cases reported, a typical postlumbar puncture headache was present during the three days to three weeks that elapsed prior to the development of the palsy.[6, 8, 10, 11, 17, 20, 21]

Large gauge Needles with Regular Bevels Were Employed in Most of the Reported Cases.—It has been shown that when large gauge needles are used, particularly those with cutting bevels, the incidence of postlumbar puncture headache is increased (see Chapter 20, page 178). Moreover, in most of the reported cases of abducens nerve palsy, 16 to 20 gauge needles with regular bevels were employed to execute the lumbar puncture. The only five cases of abducens nerve palsies in Dripps and Vandam's[10] series of 6,147 spinal anesthesias occurred in the 637 patients who were tapped with 16 gauge needles. A 16 gauge needle was also used in Brown's[8] series of 600 cases with 2 abducens nerve palsies.

Palsies Occur after Patient is No Longer in Prone Position.—Thorsén,[1] Kunkle *et al*,[3] and Wolff[33] substantiate the fact that the basic cause of a cranial nerve palsy is the decreased volume of cerebrospinal fluid in the intracranial cavity when the head is elevated

CHAPTER 24

Lesions of the Brain and/or the Intracranial Portion of the Cranial Nerves

If untoward reactions to a regional block procedure such as prolonged hypotension, convulsions or cardiac failure occur, *brain* damage may be a sequela of the oxygen deprivation which accompanies these complications. However, damage of the parenchyma of the brain seldom if ever is a result of the local anesthetic drug per se or of trauma from the placement of a needle. Even if a high spinal results during an intracranial injection with a local anesthetic agent, e.g., a gasserian ganglion block, death or brain damage should not ensue *provided resuscitative* measures are instituted immediately and continued until the physiological effects of the drug are dissipated.

On the other hand, damage to the *cranial nerves* as they course within the calvarium, while not common, has been reported a number of times as a sequela of spinal anesthesia or a diagnostic spinal tap and occasionally as a residuum from a misplaced needle during a gasserian ganglion block with neurolytic drugs, i.e., alcohol or phenol. Paralysis or paresis of any of the twelve cranial nerves or of several at once may occur.[1-3] Judging from the number of reports in the medical literature of abducens nerve involvement in comparison to the other cranial nerves, it would appear that without a doubt this cranial nerve is the one which is usually affected following spinal anesthesia.[1,21] Blatt[3] in reviewing 88 cases of cranial nerve damage found the trochlear nerve involved in 4 cases, the oculomotor nerve in 6 and the abducens nerve in the remaining 78. In the 324 cases of cranial nerve paresis reported by Thorsén[1] about 93% (299 cases) showed involvement of the abducens nerve alone. These reports indicate that the dural puncture, whether purposely or inadvertently performed, is responsible for this type of lesion. While most instances of abducens nerve paralysis have occurred following spinal anesthesia, some have been reported after diagnostic lumbar taps.[1,17,22,24] The incidence of abducens nerve involvement varies widely (see Table XXV, page 227).

As noted previously, damage to the parenchyma of the brain seldom if ever results from the actual mechanics of executing a block and reports of such damage could not be found in the literature. Therefore, this chapter deals mainly with paralysis or paresis *of the intracranial portion of the cranial nerves*. The terms "intracranial," "within the calvarium" and "cranial cavity" are stressed because damage to the cranial nerves or their branches after their exit from the skull has been considered in Chapter 11, page 112.

ETIOLOGY

The etiology of paralysis or paresis of the nerves lying within the cranial cavity with the exception of those cases which follow the injection of neurolytic drugs intracranially, has not been definitely established.[4,11,23,31] A number of factors have been cited as possible causes and while any single one of these may

tremely high concentrations are employed. However, the strongest concentration of Novocain (procaine) advocated at the present time for use in regional block is 5%, and Lundy *et al.* have shown that a 17% concentration of this drug is required to cause nerve damage if injected into the subarachnoid space of a dog.

Meningitis or Meningeal Irritation.—Masson[19] attributed abducens nerve palsy to meningeal irritation, and Bolwell[17] attributed his five cases to contaminated solutions of imported Stovaine or Tropicocaine. While there is no question that paralysis and paresis of the ocular muscles and even loss of sight may occur from a meningitis, it is unlikely that this is the mechanism following lumbar puncture unless a septic meningitis from improper technique occurs. Kennedy and Lockhart[1] point out that spinal fluids examined during the paresis in all cases they reviewed were found to be normal. On the other hand, they state that a low grade infection from some flaw in technique could be responsible for paralysis of the abducens nerve.[20]

Dattner and Thomas[23] suggest the possibility that abducens nerve paralysis may result from a latent virus infection but admit this to be "pure speculation."

Increased Intracranial Pressure.—Any increase in the intracranial pressure would tend to force the brain downward, causing it to exert traction and pressure on the cranial nerves; this has been mentioned as a possible mechanism of cranial nerve injury.[13, 16] On the other hand, Bryce-Smith and Macintosh[17] believe that the nerve may be stretched but that the lesion itself is due *to reduced, not to* the raised intracranial pressure, which Fairclough has suggested may be responsible.

Interference with Binocular Vision.—Fairclough[16] suggests that the cause of abducens nerve palsy is an *interruption* by the spinal tap or the drug used of one or more of the essentials necessary to binocular vision. This theory is not mentioned or supported by other authors reporting on cases of abducens nerve palsy.

SIGNS AND SYMPTOMS

Onset.—When the damage is caused by a *neurolytic drug*, the onset is sudden and prodromal symptoms do not occur. *On the other hand*, cranial nerve paresis or paralysis following a *puncture of the dura* alone or following spinal anesthesia usually *does not develop* for 3 to 21 days and prodromal signs and symptoms usually occur. The signs and symptoms of palsies of the cranial nerves from these latter causes are those detailed below. *They are the ones which may signal an insidious onset of an abducens nerve palsy.* Those palsies which follow the use of a neurolytic drug are a known hazard of using such an agent.

Prodromal Symptoms.—Prior to the development of the signs and symptoms of cranial nerve injury with a slow onset following puncture of the dura, the patient may *complain of symptoms resembling those which* accompany a low spinal fluid pressure following a spinal tap and including: (1) *a typical postspinal puncture headache*, (2) *dizziness*, (3) *nausea*, and (4) *a stiff neck*.

When these symptoms occur they may progress no further, but because of their potential significance they cannot be *viewed lightly*—*they indicate* that vigorous treatment to correct them must be instituted immediately. Otherwise cranial nerve damage may ensue.

Paralysis or Paresis of the Cranial Nerve or Nerves Involved.—These imply either motor and/or sensory loss of the structures the nerve supplies, depending on the type of fibers the paralyzed nerve contains. For example: (1) the acoustic nerve is entirely sensory, (2) the *abducens nerve is entirely* motor, (3) the trigeminal nerve is mainly sensory but does have a motor component, and (4) the glossopharyngeal nerve contains a relatively equal number of sensory and motor fibers.

When any of the nerves to the ocular muscles are involved, a *squint* results, and *photophobia*, *blurring of vision* and *esophoria* may ensue. Since the abducens nerve is the one affected in the greater number of cases, it is

Their conclusions are easily explained—when a patient sits or stands the brain sags because of the effect of gravity and if the cushioning effect of the cerebrospinal fluid is no longer present due to leakage it is reasonable to assume that excessive traction or pressure on the cranial nerves will result

Anatomy of the Cranial Nerves—Most of the cranial nerves are subject to pressure or traction if the cerebrospinal fluid is reduced The trochlear nerve being the most slender and having the longest intracranial course might perhaps be thought to be the most vulnerable but on the contrary the abducens nerve is more vulnerable because (1) its course is also long (2) it makes a sharp bend over the angular apex of the petrous temporal bone through Dorello s canal and (3) as it passes between the pons and occipital bone it is crossed by the anterior inferior cerebellar and the internal auditory artery [16 34 3] These obstacles predispose to its compression when hypotension of the cerebrospinal fluid occurs

Damage to Blood Supply to Nucleus of the Sixth Nerve and Hypoxia or Anoxia—The blood is supplied to the abducens nucleus via two very narrow arteriae medianae from the basilar artery and it is possible (though unlikely) that a vascular accident or *any prolonged hypotension* during the surgical procedure or in the postoperative period might result in hypoxia or anoxia of the nucleus and consequently in palsies of the abducens nerve [3]

While Dattner and Thomas [3] believe this to be unlikely the theory is supported somewhat by the fact that cranial nerve palsies have occurred in the absence of dural puncture Woltman [36] has cited two cases of abducens nerve palsy subsequent to ether anesthesia Courville [37] has reported several cases of cranial nerve palsy (abducens nerve included) following inhalation anesthesia and Norman [43] noted a case of partial right third cranial nerve palsy following anesthesia with Pentothal d tubocurarine chloride cyclopropane and oxygen In these cases the complication could have been caused by anoxia

Neurolytic Drugs—Occasionally the gasserian (semilunar) ganglion is injected with *alcohol* or *phenol* as a therapeutic measure to relieve the pain of trigeminal neuralgia (tic douloureux) If the needle point is misplaced and lies outside the capsule of the ganglion cranial nerve palsies may ensue, particularly those of the oculomotor the trochlear nerve the abducens nerve and the glossopharyngeal nerve

Local Anesthetic Agents—The direct action of the local anesthetic agent on the cranial nerves or their nuclei has been suggested as a possible cause of palsy [1-] It is possible that this might have been a factor in the early days of spinal anesthesia when drugs for spinal anesthesia were in their developmental stages Spielmeyer [38] in his article on Stovaine points out that if nerve cells are affected by anesthetic agents at all it is most likely that of the cranial nerves the abducens nerve would be involved since there are fewer cells in its nuclei The hypothesis that local anesthetic agents may act directly on the cranial nerves may also be somewhat supported by the consideration of Kennedy and Lockhart [0] who note the superficial relationship of the abducens nerve to the fourth ventricle and write "It must be remembered also that the abducens nerve has a superficial relation to the fourth ventricle which communicates with the subarachnoid space into which the anesthetic agent is injected

However with the time tested low toxicity local anesthetic agents in use today it is highly unlikely that the toxicity theory is a significant etiological factor in cranial nerve palsy In the cases of abducens nerve palsies which have recently occurred no one local anesthetic agent can be singled out for scrutiny since Pontocaine Novocain and Nupercaine have all been employed [6 8 11 1] Moreover it is difficult to condemn these drugs or any others in light of the fact that cranial nerve palsy has occurred following diagnostic lumbar punctures where no drug at all has been injected into the subarachnoid space [1 17 4]

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Perhaps the only time that the local anesthetic agent might be condemned is when ex

an abdominal binder and if necessary *epidural (peridural)* injections of saline via an indwelling catheter (see Chapter 20 page 190)

Prevent Atrophy of Muscles—Since the palsy is only temporary in most cases, the muscles involved should be stimulated with weak galvanic current to prevent atrophy from disuse

Consult an Ophthalmologist—When the ocular muscles are involved, as they are in the greater number of cases a specialist should be consulted Usually he will cover the eye to eliminate the distressing diplopia However no attempt to correct the squint will be made until the lesion has persisted without improvement for at least a year[41]

Prognosis—Most cranial nerves affected by paresis will as with the usual peripheral nerve injury, return to normal function Those which occur from lumbar punctures or spinal anesthesia particularly abducens nerve palsy usually resolve in one week to four months From their own experiences and a review of the literature Kennedy and Lockhart[20] concluded that 90% of the cases of abducens nerve palsy following spinal anesthesia recover in 8 weeks or less If the palsy results from the use of a neurolytic drug nine months to one year may be necessary for recovery

In spite of the fact that most cases of cranial nerve palsy recover, the prognosis must be guarded as Hingson *et al*[9] report a case of abducens nerve palsy of five years duration with no improvement Thorsén[1] also cites cases of permanent paralysis

COMMENTS

Lumbar Puncture Death—This is due to damage to the vital centers in the medulla It is most likely to occur with the patient in the sitting position To date this type of problem has not been encountered at the Mason Clinic On this subject Maxson[40] warns that "By careless and rapid withdrawal of large quantities of spinal fluid through a large needle with the patient in the sitting posture the normal water cushion of the brain is removed and herniation of the brain stem through the foramen magnum may occur In such a case death is due to pressure on the medullary centers and may be sudden Most of the recorded cases had intracranial tumors others cerebral hemorrhage A spinal anesthesia should never be attempted in such cases and lumbar puncture if performed should be done in the lateral posture A small caliber needle is a safeguard"

Cranial Nerve Palsy Following the Use of Trichlorethylene (Trilene, Trimar)—At the present time trichlorethylene is being used extensively as an analgesic during obstetrical labor Often obstetrical patients are given a spinal anesthetic just before delivery Should a cranial nerve palsy ensue the physician might condemn the regional block procedure It must be emphasized that trichlorethylene was originally employed to treat trigeminal neuralgia because of its depressant action on the cranial nerves This depressant action on the nervous tissue may result in cranial nerve paralysis particularly if the drug is used over a prolonged period, if it has started to decompose or if it is given in a closed system Twenty two incidences of cranial nerve palsy following the use of this drug in anesthesia have been cited[4] In addition to these 22 cases 2 cases of facial nerve palsy following the use of Trilene for labor and delivery were described in a recent letter to the author

If cranial nerve palsies following the use of trichlorethylene do not clear within a month they may be permanent or at least semi permanent, i e, last 6 months or longer[4] While the trigeminal and facial nerves are the ones usually involved palsies of all the cranial nerves with the exception of the first second, ninth and eleventh nerves have been reported following the use of trichlorethylene[4]

Convulsions and Retinal Hemorrhage Following "Pressure Caudal—Not infrequently the anesthesiologist is requested to perform pressure caudal injections for sciatic or back pain This entails the rapid *injection of* 50 to 90 cc of solution into the caudal canal The solution must be injected rapidly so that it will not escape through the *foramina* and thus

interesting to note that (1) its incidence is greater in women than in men (2) the ratio of unilateral to bilateral palsy is 3 to 1 and (3) neither eye is predominantly involved[11] Furthermore, the affection may develop on the side opposite the one on which the patient is lying at the time a large amount of spinal fluid is lost because the brain sags toward the side which is down, placing the greatest tension on the opposite nerve Bryce Smith and Macintosh[17] cite such a case following the administration of a spinal block

PROPHYLAXIS

Prophylaxis to a large extent depends on whether a neurolytic drug is to be injected or a spinal puncture performed

Prevention of the Incorrect Placement of Neurolytic Drugs During a Gasserian Ganglion Block—When neurolytic drugs are to be injected into the gasserian (semilunar) ganglion the only prophylactic measures which may assure correct placement of the needles are (1) x ray studies of the placement of the needle (2) injection of a Diodrast solution equal to the amount of neurolytic agent to be used followed by roentgen studies within 2 to 5 minutes to determine whether the solution remains within the capsule of the ganglion or spreads diffusely and (3) injections of small amounts of local anesthetic agents and study of their effects prior to the injection of the neurolytic drug

Even when these precautions are taken it is possible that cranial nerve damage may result from a change in the needle s position due to (1) movement by the patient due to pain which accompanies the injection of a neurolytic drug or (2) slight changes in the patient s position during the interval between the taking of the x rays and the decision to inject the neurolytic drug

Recently Jaeger[44] has suggested that the gasserian ganglion be blocked (coagulated and destroyed) with boiling distilled water as a means of avoiding the dangers involved in the use of neurolytic drugs He states "It is improbable that damage to the brain or other cranial nerves will result if no more than 1 ml of water is injected at any one time since the water temperature is immediately lowered to a safe level the instant it is diluted by the intracranial cerebrospinal fluid"

Prevention of Cranial Nerve Palsies Following a Spinal Tap—As Greene et al [1] state "Prevention of postpuncture palsies of the cranial nerves begins with the prophylaxis of the lumbar puncture headache This prophylaxis consists of (1) *use of a small gauge needle,* (2) *use of a Greene or pencilpoint needle* (3) *hydration and replacement of blood loss* and (4) *bed rest etc* (see Chapter 20 page 183) If a postpuncture headache should develop even though some or all of these steps have been observed it should not be considered in a casual manner but treated vigorously as advocated in Chapter 20 page 190 *It must always be remembered that this type of headache may be the forerunner of a cranial nerve palsy*

Greene *et al*[21] believe that the most effective and prompt means of removing the threat of palsy is recumbency, but this is not necessarily true It is granted that bed rest will prevent the downward pressure, but as the patient lies down and the brain sags upward traction in a different direction may develop The case cited by Bryce Smith and Macintosh[17] and mentioned above is a good illustration

The prophylactic use of nicotinic acid to dilate the vessels of the brain and increase the production of spinal fluid by the choroid plexus is questioned by Greene *et al*[21] on the basis that its action increases the volume and relative weight of the brain and therefore aggravates the traction or pressure Whether or not this is an important consideration only further experimental studies can determine

TREATMENT

As soon as palsy or paralysis is noted the following therapy should be instituted

Correct Headache—If the headache is still present when the palsy occurs it should be vigorously treated with *bed rest, hydration*

an abdominal binder and if necessary *epidural (peridural)* injections of saline via an indwelling catheter (see Chapter 20, page 190).

Prevent Atrophy of Muscles.—Since the palsy is only temporary in most cases, the muscles involved should be stimulated with weak galvanic current to prevent atrophy from disuse.

Consult an Ophthalmologist.—When the ocular muscles are involved, as they are in the greater number of cases, a specialist should be consulted. Usually he will cover the eye to eliminate the distressing diplopia. However, no attempt to correct the squint will be made until the lesion has persisted without improvement for at least a year.[41]

Prognosis.—Most cranial nerves affected by paresis will, as with the usual peripheral nerve injury, return to normal function. Those which occur from lumbar punctures or spinal anesthesia, particularly abducens nerve palsy, usually resolve in one week to four months. From their own experiences and a review of the literature, Kennedy and Lockhart[70] concluded that 90% of the cases of abducens nerve palsy following spinal anesthesia recover in 8 weeks or less. If the palsy results from the use of a neurolytic drug, nine months to one year may be necessary for recovery.

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Convulsions and Retinal Hemorrhage Following "Pressure" Caudal.—Not infrequently the anesthesiologist is requested to perform pressure caudal injections for sciatic or back pain. This entails the rapid injection of 50 to 90 cc. of solution into the caudal canal. The solution must be injected rapidly so that it will not escape through the foramina and thus

fail to elevate the pressure in the epidural space

Following the injection of the solution it is common for the referring physician to manipulate the patient's legs and back. This is usually performed with much vigor. Because the manipulation of the back is attended with pain the patient must receive either a regional anesthetic or a general anesthetic. Following regional anesthesia we have observed convulsions not due to systemic toxic reaction. Following general anesthesia and this technique Callahan, McConville and Goss[45] have observed 8 cases of retinal hemorrhage.

Convulsions.—To perform a pressure caudal and back manipulation it has been our custom at the Mason Clinic to (1) place the needle in the caudal canal, (2) inject 30 cc. of 1% Xylocaine solution, (3) wait 15 minutes for the anesthetic effects of the Xylocaine to become well established, (4) then inject 50 to 70 cc. of normal saline rapidly until the patient complains of either an aching between the scapula or a headache, and (5) vigorous manipulation of the back and legs.

On a number of occasions after 30 to 40 cc. of the saline had been injected and before the complaint of headache, the patient had a convulsion which lasted 10 to 20 seconds. Presumably this is due (1) to motion of the cord caused by the rapid injection of the saline or (2) to the increase in the spinal fluid pressure which results from filling the epidural space, which then forces the cerebrospinal fluid from the dural sac into the cranial cavity, increasing intracranial pressure. To date we have seen no untoward effects from either the injection, the convulsion, or the manipulation.

These patients are never given general anesthesia prior to such an injection, and whenever the patient complains of pain between the scapula, headache, or has a convulsion, the injection is suspended, but the manipulation is performed.

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fail to elevate the pressure in the epidural space

Following the injection of the solution it is common for the referring physician to manipulate the patient's legs and back. This is usually performed with much vigor. Because the manipulation of the back is attended with pain the patient must receive either a regional anesthetic or a general anesthetic. Following regional anesthesia we have observed convulsions not due to systemic toxic reaction. Following general anesthesia and this technique Callahan, McConville and Goss[40] have observed 8 cases of retinal hemorrhage.

Convulsions.—To perform a pressure caudal and back manipulation it has been our custom at the Mason Clinic to (1) place the needle in the caudal canal, (2) inject 30 cc. of 1% Xylocaine solution, (3) wait 15 minutes for the anesthetic effects of the Xylocaine to become well established, (4) then inject 50 to 70 cc. of normal saline rapidly until the patient complains of either an aching between the scapula or a headache, and (5) vigorous manipulation of the back and legs.

On a number of occasions after 30 to 40 cc. of the saline had been injected and before the complaint of headache, the patient had a convulsion which lasted 10 to 20 seconds. Presumably this is due (1) to motion of the cord caused by the rapid injection of the saline or (2) to the increase in the spinal fluid pressure which results from filling the epidural space which then forces the cerebrospinal fluid from the dural sac into the cranial cavity, increasing intracranial pressure. To date we have seen no untoward effects from either the injection, the convulsion or the manipulation.

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CHAPTER 25

Miscellaneous Complications from Paralysis of the Autonomic Nervous System

Other than hypotension there are a number of complications which have been reported from paralysis of the sympathetic portion of the autonomic nervous system. These have mainly occurred following spinal and epidural block procedures but may appear following peripheral nerve blocks of the splanchnic (celiac) plexus or of the lumbar sympathetic ganglia. From the number of reports in the literature it can be assumed that the complications discussed in this chapter are infrequent. This is fortunate since they are usually of a serious nature.

PULMONARY EMBOLISM

Pulmonary embolism during or shortly following spinal, epidural and lumbar sympathetic blocks has been reported.[1-5]

Etiology.—According to Owens and Smith[1] when one part of the body's circulatory system is dilated by a block another part must constrict to compensate for this shift in blood volume. Discussing the application of this "borrowing lending" theory (hemometakinesia) proposed by DeBakey *et al.*[6] as it applies to lumbar sympathetic block, Owens and Smith[1] state: "Following a lumbar sympathetic block there is a marked vasodilatation in the lower extremity associated with increase in blood volume and rate of blood flow. Here we have blood 'borrowed' by the leg at the expense of the remainder of the body as indicated by cooling and decrease in volume of pulse deflection and total volume of these areas [rest of body] as noted on a plethysmograph. Not all of the expense of this shift of blood is borne entirely by the opposite leg, since its decrease is not as great as the other leg's increase. Here every organ that can afford to lend blood does so and the amount depends on the quality and quantity of the shift."

When this "borrowing lending" is caused by a regional block procedure in the elderly patient with arteriosclerotic heart disease, Owens and Smith believe that the patient is in a precarious position. If a pulmonary infarction develops at this time it would probably result in death. The combination of pre-existing heart disease, reduction of blood supply to the heart by the block, and additional myocardial insufficiency produced by the spasm from the pulmonary embolus are more than such a patient could survive.

Further, they feel that in treating thrombophlebitis excessive manipulation or movement of the blocked leg or legs together with the dilatation from the block facilitates the release of a thrombus or the breaking off of an embolus. One would conclude from this that a patient should not be permitted to be ambulatory immediately following a lumbar sympathetic block—a hypothesis which clinical experience has not entirely substantiated, for often this block and early ambulation is one of the treatments used for thrombophlebitis.

Signs and Symptoms.—The signs and symptoms of pulmonary embolism are well known to physicians; the cardinal ones are: (1) pain in the chest, (2) oxygen want, (3) pulmo-

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PHLEBOTHROMBOSIS AND THROMBOPHLEBITIS

Allen Barker and Hines[1] believe that from a clinical standpoint no differentiation between thrombophlebitis and phlebothrombosis should be made as it will only result in confusion By definition phlebothrombosis is partial or complete venous occlusion by an intravascular clot which is unassociated with inflammation the clot being loosely attached to the vein wall[13] On the other hand thrombophlebitis is partial or complete venous occlusion by an intravascular firmly adherent clot which is associated with and dependent upon inflammation of the vein wall[13] Ochsner and DeBakey[13] feel that phlebothrombosis may exist for a short time prior to the onset of thrombophlebitis

Etiology and Prophylaxis—While the author and his associates have not observed phlebothrombosis attributable to a regional block Wasmuth[14] states Phlebothrombosis is defined as the intravascular coagulation of blood During anesthesia the occurrence of phlebothrombosis follows the retardation of blood flow either by external pressures or the suppression of circulation by other means Spinal anesthesia with the peripheral vascular paralysis of the lower extremities frequently lays the groundwork for thrombosis if precautionary steps are not taken to alleviate these dangers Elastic bandages applied snugly around the legs prevent the pooling of blood in the venous system by constant external pressure Other prophylactic steps are (1) avoid depression of circulation especially by hypotension (2) prevent pressure at dangerous points such as the popliteal space and (3) prevent stretching of the limbs with concomitant stretching of vessels

"The use of elastic bandages on the legs is not without danger Their efficiency in preventing phlebothrombosis from stagnation of blood is great However during bouts of hypotension the external pressure applied to the leg may become greater than the arterial pressures Long periods of ischemia of the peripheral tissues are to be avoided"

Treatment—The treatment of phlebothrombosis and thrombophlebitis following regional block procedures is the same as that which follows the disease from other causes and consists of anticoagulants fluid therapy surgical ligations antibiotics sympathetic block etc

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PART III

INCIDENTAL COMPLICATIONS

Introduction to Part III

THIS PART of the book discusses those complications which occur during or following a regional block procedure. They are not definitely a direct result of an untoward pharmacological action of the drugs used, tissue damage from lytic drugs or trauma associated with the technique. *They may be directly or indirectly associated with the block procedure and may occur following local infiltration, peripheral nerve block, and spinal or epidural block.*

CHAPTER 26

Broken Needles or Catheters

The breakage of a needle or a catheter may be of little significance other than to create a momentarily embarrassing situation provided the part of the needle or catheter remaining in the patient is easy to retrieve Unfortunately this is not always the case During the past fourteen years the author has had four needles and two plastic catheters for *continuous anesthesia break in patients* surgery being required for their extraction Of the 4 needles one broke while giving a single dose caudal with a non malleable 19 gauge needle one while inserting a needle for a transsacral block another while seeking paresthesias for a brachial plexus block and the fourth 2½ hours after a continuous caudal anesthetic with a malleable needle had been established and three doses of the local anesthetic drug had been given with success (see Figure 46 page 243) The two plastic epidural catheters had been in place in the epidural space for approximately 72 hours in an attempt to effect a prolonged sympathetic block when they broke The break occurred where they entered the skin and the broken end then submerged into the subcutaneous tissue (see Figure 47 page 243)

ETIOLOGY

Focal Weak Spots—When a needle or catheter breaks it is usually caused by stress being placed on a *known* or *unknown* weak spot In the case of needles focal weak spots usually occur at the point where the shaft of the needle is fitted to the hub or where a bent needle has been straightened In the case of plastic catheters we have found that the focal weak spot is usually where the catheter *emerges from the skin*

In the needle which has a focal weak spot breakage may result even when the needle is handled correctly but is more prone to occur when it is forced against bone or when an attempt to change its direction is made without first withdrawing the point to the subcutaneous tissues

Incorrect Method of Inserting Catheter—In any continuous regional block technique the shearing off of a plastic or ureteral catheter by trying to withdraw the catheter from the needle once the catheter has passed the tip of the needle is an inherent danger of this technique[1]

PROPHYLAXIS

Breakage of a needle or catheter may not be the fault of the physician but may result when the patient is turned or moved This appeared to be the cause in the one case mentioned in which a continuous caudal needle broke The same cause must have been operative in breaking the two plastic catheters which had to be removed by surgery since similar catheters taped in place in the same fashion have not broken although they have been left in place for seven days Nevertheless the following precautions will prevent or minimize the breaking of needles or catheters in the patient

Check All Needles and Catheters Carefully—Prior to use all needles and catheters should be carefully examined for weak spots and the stylet checked to see that it is freely movable A stylet which is stuck in a needle



Figure 46 Broken malleable caudal needle (A) Anterior posterior view (B) Lateral view

may indicate a rusted spot in the needle while one which is difficult to move in a catheter may indicate that the catheter has been sterilized with a sharp bend in it

Avoid Kinking a Catheter — A catheter should not be kinked during sterilization This may be avoided by sterilizing catheters in Petri dishes (see Figure 48 page 244) or transparent cellophane tubes[2,3] When a catheter is taped in place care should be taken to see that it does not turn at an acute angle as it enters the skin

Do Not Straighten Bent Needles —Once the shaft of a needle has been bent it must be discarded not straightened and reused To sterilize and reuse such a needle is to ask for trouble The great majority of needles in use today will rust when their polished surface has once been damaged and this rusting results in a focal weak spot

Use Security Bead Needles —Since most needles which break during field block local infiltration and nerve block break at the junction of the hub and shaft the use of security bead needles will prevent the physician from accidentally inserting the needle to that point Thus if a security bead needle does break at the junction of the shaft and the hub it is easily removed (see Figure 49 page 244)

Handle Needles Correctly —Never should a needle be forced against a bony landmark (see Figure 50 page 245) Likewise its shaft should never be bent when changing its direction (see Figure 51 page 245)

Do Not Withdraw Catheter Once It Has Passed the Tip of the Needle —When catheters are used they must be carefully inserted and once the catheter has passed the tip of the needle it must never be withdrawn while the needle remains inserted or the catheter may be sheared off[1] If the catheter meets an obstruction and cannot be advanced after it has passed the point of the needle then both the needle and catheter should be withdrawn simultaneously and the procedure started anew

Do Not Reuse Needles Indefinitely —Re

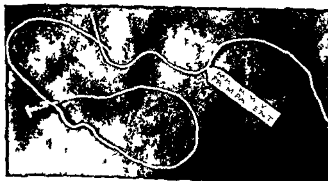


Figure 47 Broken plastic catheter

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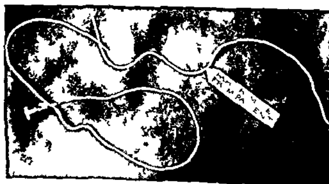


Figure 47 Broken plastic catheter

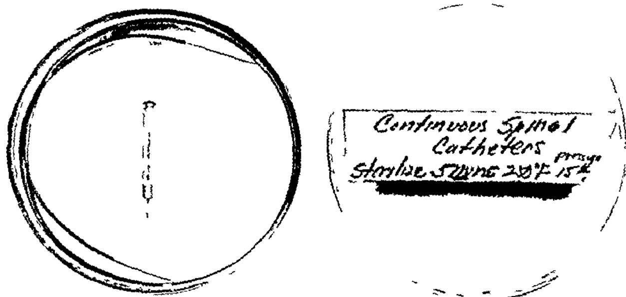


Figure 48 Petri dish used to sterilize catheters and avoid their kinking

Figure 49 Needle with security bead broken at junction of hub and shaft while doing a brachial plexus block Removal was made easy by security bead

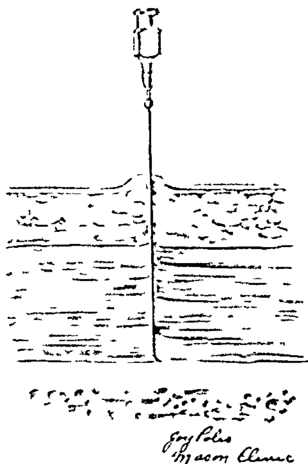


Figure 50 Needle point bent on bone by too rapid insertion of the needle when seeking bony landmarks

sterilization of needles tends to make them lose their temper and become brittle Needles also tend to dull from continued use Therefore it has been our policy to change all needles in our regional block trays at least once a year

TREATMENT

Whenever a needle or catheter breaks every effort to recover it including surgery must be made immediately A broken needle is singularly dangerous because it has the tendency to change positions i e migrate as the patient moves and since both its point and the broken edge are sharp it may puncture a vital organ The use of x ray and the placing of guide needles as well as the use of the Berman Locator prior to and during surgery may prove of aid

Lahey [4] described a method of recovering broken spinal needles without the use of x ray or the Berman Locator which has proven satisfactory at the Lahey Clinic It consists primarily of exposing the aponeurosis over the erector spinae splitting it on each side of the spine palpating on each side of the spine with the index finger until the broken needle is found and removing it with a right angled hemostat Should the broken needle lie directly between and beneath the spinous process the interspinous ligament is gently grasped with a right angle (gall bladder type Mixter) forceps and upward traction exerted on this ligament until the needle is found by palpation by surgical exploration of the ligament, or both

To recover the broken needles and catheters in all but one of our cases required only an incision in the skin and exploration of the subcutaneous tissues One case required more

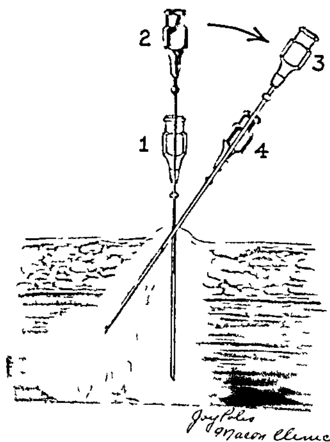


Figure 51 Correct method of changing direction of the needle (1) Needle deep in tissue (2) Needle withdrawn to subcutaneous tissue (3) Direction of needle changed (4) Needle reinserted If the direction of a needle is changed in this fashion its shaft will not be bent

surgery and the bone plate over the caudal canal had to be cut away by a ronguer to recover the broken part of the needle

COMMENT

Possible Loss of the Tip of the Whitacre (Pencilpoint) Point Needle—The tip of the Whitacre point needle is soldered to its shaft and according to Schwidetzky may loosen after repeated sterilizations He feels that when this occurs the point could possibly be left in the patient s back He postulated that the soldered junction could become loose be cause the expansion coefficients of the metal in the point and in the shaft are different No instances of such breakage could be found in the literature and Whitacre[6] has told the author that no such complication has ever arisen in his series of cases

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Unsatisfactory Anesthesia

In the true sense of the word this is not a complication of nerve block since usually no untoward sequelae are caused by it. Nevertheless, inasmuch as it does occur, it should perhaps be noted that when blocks are unsatisfactory, the cause in most instances is due to technical errors on the part of the physician performing the block. *Seldom is the failure caused by the deterioration of a local anesthetic agent or by anatomical anomalies.* The over-all incidence of complete failure of regional block techniques in the hands of physicians skilled in their usage is approximately 5 to 7%.

Often intravenous Pentothal (sodium thiopental) and/or intravenous fluids are administered when either the regional block analgesia is unsatisfactory, the patient is restless or the surgery is prolonged. The two most common local complications of administering intravenous Pentothal and fluids are:

Vascular Spasm Following Intra-arterial Administration of Pentothal.—Often Pentothal analgesia is given intravenously in conjunction with regional block analgesia. If the Pentothal is accidentally started in an artery the patient will complain of severe pain in the extremity. When this occurs the physician should immediately suspect an intra-arterial injection but he should not remove the needle until he has slowly administered 50 to 100 cc of 0.2% Novocain (procaine) through it to relieve the spasm. If this is not successful in relieving the spasm or if the physician neglects to do it, then either lumbar sympathetic or stellate ganglion block therapy (depending on whether the leg or arm is involved) should be instituted immediately to relieve the vasospasm and prevent further sequelae.[1]

Swelling from Extravasation of Intravenous Fluids During Surgical Procedure.—When intravenous fluids are given during surgery, the needle may become dislodged from the vein, resulting in a large extravascular fluid deposit in the extremity and pain. If the tumefaction is not treated immediately in the patient with arteriosclerosis or other vascular diseases, extravasation may cause tissue slough, particularly if the solution contains a vasoconstrictor drug or Pentothal. This type of complication may be prevented by injecting 10 to 20 cc of either a normal saline solution or a 1.0% Novocain solution or a 0.15% Pontocaine solution containing 150 to 300 T.R.U. of hyaluronidase (Wydase, Alidase, Diffusin) into and around the tumefaction, gently massaging the area and applying an elastic (Ace) bandage for a few hours.[2] If this does not suffice, then sympathetic block therapy should be initiated.

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CHAPTER 28

Amnesia in Outpatients Following Regional Block Procedures

AMNESIA in outpatients during regional block procedures may result either from premedication or from the drug used. In the hospital patient amnesia is usually sought but in the outpatient it may lead to difficulty.

Premedication.—Outpatients should receive little or no premedication and if they are medicated they should stay in a recovery room for two to four hours and then be escorted home. If these precautions are not followed the patient may sustain an injury on the way home which may result in a law suit. Barbiturates in particular are known to exert a prolonged effect and cause impairment in cerebration. Von Felsinger *et al.*[1] state: "The effect of 0.1 gm. of pentobarbital sodium on a variety of complex psychological functions has been investigated. As long as five and one half to eight hours after ingestion of the drug and following a light breakfast significant impairment of visual perception, attention, arithmetical performance and recall was demonstrated. Associations were increased in number after drug administration but these were less reality determined, i.e., showed less relation to external stimuli than after the placebo. In a confusing and disturbing test situation the drug effect was to facilitate resistance to distraction. Serial learning and an analogies test failed to show drug effect.

"These findings are offered as objective confirmation of the frequent complaint of 'hang over' after the use of pentobarbital sodium in ordinary hypnotic dose. It is suggested that the prolonged effects detected here must be considered as factors which limit the usefulness of this and similar agents when such persistence of effect might be undesirable, that is, where it is important that the individual's full mental faculties be available."

During World War II this action of the barbiturates was drawn to the author's attention by a case of a medical officer to whom Pentothal anesthesia was administered for the opening of a furuncle at eight o'clock in the morning. This officer, after what appeared to be a full recovery from the effects of the Pentothal, performed an operation at eleven o'clock and following surgery went about performing his usual duties which included writing orders until five o'clock in the afternoon when he drove home. The next morning in surgery he volunteered the information that he could not recall his activities of the previous day including his drive home. The prolonged effects of Pentothal are further substantiated by Dundee's[3] research in both humans and dogs.

Local Anesthetic Drugs.—In general local anesthetic drugs do not cause amnesia. However, Hickey,[2] an anesthesiologist from Little Rock, Arkansas, informed the author that he knew of twelve dental patients who had received Xylocaine (lidocaine) in dentists' offices and were unable to remember facts and events which transpired in the five hours following the execution of the block. Xylocaine is known to have a depressing effect on the brain when given in dosages exceeding 300 mg. (see Chapter 1, page 16). However, it was a bit surprising to find that small amounts of the drug such as those commonly used in

dentistry, i.e. 2 to 5 cc. of 2% solution (40 to 100 mg.) may have the same effect.

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Retention of Urine

THE INABILITY of a patient to urinate following anesthesia of any type is not infrequent[1,2,3] Its incidence is greater following pelvic surgery and surgery of the urogenital system than following upper abdominal surgery or surgery other than intra abdominal

ETIOLOGY

Urinary retention is usually attributed to *pre existing urinary infection or difficulty* the anesthetic the horizontal position of the patient the pain of injured tissues the surgical procedures and drugs such as opiates[2,4] When spinal or epidural blocks are used most surgeons almost inevitably blame the anesthetic procedure Bellis states "Spinal anesthesia temporarily establishes a type of neurogenic vesical dysfunction, probably due to the anesthetic agent The bladder wall remains somewhat insensitive to its distending contents and although the expulsive force of the detrusor may actually not be diminished the normal strong reflex vesical contractions which coincide with desire to void, do not appear until the volume of the bladder and the intravesical tension are so great as to invite further pressure anesthesia of the wall, and a continued sensory type of retention which initiates urinary infection" On the other hand Bomze in a recent article reviewing 121 cases 25 of which had difficulty in voiding postoperatively states "The presence of findings in the admission urinalysis the presence or absence of urinary complaints preoperatively, and the type of anesthetic used had no bearing on the occurrence or severity of postoperative urinary dysfunction" Of these 25 patients 9 received spinal anesthesia and 16 had general anesthesia

It has been our conclusion at the Mason Clinic that the two main reasons for the failure to urinate following a surgical procedure relate to the type of surgery performed and hypotension

Type of Surgical Procedure —When pelvic surgery is performed it has been observed that the incidence of urinary *retention* increases irrespective of the type of anesthetic Bomze[2] substantiates this observation by claiming that "The important fact demonstrated [by his study] is that when the surgery involved any great amount of handling of the bladder or dissection about the bladder the patient was very likely to have postoperative bladder dysfunction"

Hypotension —If a patient has a low blood pressure or blood volume postoperatively production of urine may be reduced because filtration pressure is inadequate

SIGNS AND SYMPTOMS

When urinary retention occurs the bladder becomes distended and although the urgency to urinate is present, the patient cannot void Pain may be quite severe until the patient successfully voids or is catheterized

PROPHYLAXIS

Atraumatic Surgery —According to Bomze[2] the best prophylaxis of postoperative urinary retention is to avoid injury to the bladder during operation

Use Indwelling Catheter.—If trauma to the bladder occurs at operation an indwelling catheter should be used for the first 24 to 48 hours or longer, depending on the degree of trauma. It is preferable to do this rather than to leave orders to catheterize the patient every 8 to 12 hours. The indwelling catheter puts the bladder at complete rest and allows the edema caused by surgical trauma to subside.

Bellis[5] further emphasizes the importance of catheterizing the patient before over distention of the bladder occurs. He states "To wait until the bladder is distended to these levels [500 to 1300 cc —volume necessary to initiate sensation to void in patients after *spinal anesthesia is dissipated*] is to court infection. If malaise chills fever and leukocytosis follow catheterization after the bladder has been so distended the blame need not be placed on the catheter. The surgeon must call himself to task for allowing the *seeds of infection to become implanted in the* bladder wall by permitting distention pressure necrosis, retention and reduced sensitivity to pursue their vicious cycle. Early catheterization of patients after administration of spinal anesthesia and perhaps after in*halation anesthesia is a necessary therapeutic* adjunct.

"The surgeon must be mindful of the hour at which the bladder was emptied preoperatively the diuretic effect of the sympathetic paralyzing anesthetics and the fluids administered postoperatively. *Often a patient will* require catheterization 4 or 5 hours after returning from the operating room where operation perhaps of 1 hour has been performed and following which intravenous solutions have been administered. Merely to direct a *subordinate to* catheterize in 12 hours hours if patient has not voided is a fateful and foreboding procrastination. Early catheterization by preventing pressure anesthesia of the bladder wall, will facilitate early subsequent spontaneous voiding while delayed or widely spaced catheterization *serve to* aggravate the neurogenic dysfunction and precipitate infection."

Avoid Postoperative Hypotension.—Blood and fluid replacement is essential during surgery and if necessary, vasoconstrictor drugs should be used during the postoperative period to maintain the blood pressure until the regional block procedure has dissipated itself.

Preoperative Training in Use of Urinal and Bedpan.—Collins[3] states, "It is difficult to understand however why the anesthesia and spinal in particular as well as type of operation should be exclusively emphasized as the cause of retention when the majority of patients cannot use a urinal in bed preoperatively. I believe that patients should be encouraged to use and be taught to use a urinal and bedpan preoperatively. In my experience such a regimen has significantly diminished postoperative retention."

TREATMENT

The treatment of urinary retention depends *to a great extent on whether or not the bladder* has been traumatized during surgery.

Bladder Not Traumatized.—In these cases the bladder dysfunction is probably neurogenic i.e. either the trigone does not contract or there is a decrease in tone of the detrusor *muscle and the treatment of choice consists* of one or two catheterizations following which the patient usually voids voluntarily.

Voluntary urination may be encouraged by hot packs to the perineum standing or sitting the patient in the upright position early ambulation and/or suggesting another catheterization in the patient's presence. Prostigmine (neostigmine) physostigmine (eserine) Urecholine (bethanechol) and Mecholyl (methacoline) have been advocated as urinary stimulants in cases which are primarily neurogenic i.e., those resulting from block anesthesia where the bladder has not been traumatized. But Creevy[4] points out that measures which rely on cholinergic vagotropic, or parasympathomimetic drugs audible running water etc to produce voiding do not produce the *physiological emptying* permitted by a correctly passed well lubricated catheter. Bellis[5] stresses that urine obtained by means other

than a catheter is more often than not merely overflow, i.e. the bladder is not emptied below the normal capacity.

Whenever prostigmine or physostigmine is employed the possibility of a toxic reaction, while relatively rare, must be considered. At the Mason Clinic one such case has been seen when prostigmine was used for a reason other than retention and Richardson[6] reports another. Goodman and Gilman[7] describe reactions to these drugs as follows: "The symptoms and signs of poisoning by physostigmine and prostigmine are sufficiently similar to allow a single description to suffice for both drugs. Poisoning from these alkaloids usually occurs *accidentally in medical practice*. Symptoms come on quickly after ingestion of the drug and soon reach their peak. The effects on the bowel are as a rule first to be noted and violent peristalsis, nausea, vomiting, colic, persistent purging and vomiting occur. The skeletal muscle phenomena consist of fibrillary twitchings all over the body, nystagmus and dysarthria. Restlessness and weakness are prominent. The pupils are pin point in size and distant objects are blurred. Sweating, salivation and lacrimation may be marked. Dyspnea is present due to bronchiolar constriction and abundant pulmonary secretions. There is urinary urgency and difficulty in voiding. The skin is ashen gray in color and bathed in a cold perspiration. The heart rate is rapid, the pulse weak and the blood pressure at shock level. Consciousness is not disturbed except for faintness and the fear of impending disaster. A fatal outcome is usually caused either by pulmonary edema or central respiratory paralysis. Death may occur within one half to two hours after symptoms are first noted.

"Treatment consists of hypodermic or intravenous administration of one or two mg. of atropine sulfate dependent upon the urgency of the case. Atropine quickly counteracts the serious effects of physostigmine, namely those on the lungs and circulation, but it does not influence the annoying but innocuous skeletal muscle twitchings. These subside as the drug is eliminated from the body."

Bladder Traumatized.—When retention is due to this cause the treatment must be twofold: first, further damage to the bladder must be prevented, and second, residual urine should be avoided. To prevent overdistention of the bladder with further trauma and to prevent residual urine from accumulating the bladder should be kept empty and at rest by means of an indwelling catheter. The length of time the catheter remains in place depends on the speed with which the bladder recovers *and may be 4 to 6 days*. While Urecholine, Mecholyl and prostigmine may be used when the bladder is not traumatized, Bomze[2] emphasizes that they are not effective in cases where the bladder is traumatized.

When an indwelling catheter is used all precautions to avoid infection of the bladder should be observed, i.e., connectors, rubber tubing and drainage bottle should be sterile. The urine should be examined daily by the laboratory for bacteria. If infection occurs it must be treated by the usual antibiotic therapy.

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CHAPTER 30

Atelectasis and Bronchopneumonia

ATELECTASIS and bronchopneumonia seldom if ever occur when regional block procedures are employed for extremity or extraperitoneal surgery However following intraperitoneal operations these two lung complications occur at about the same frequency under regional block procedures as under general anesthesia

ETIOLOGY

The anesthetic agent or technique per se is usually not the cause In most instances atelectasis and bronchopneumonia are caused by one or more of the following (1) the preoperative status of the patient (2) too heavy sedation (3) pain from surgery and (4) failure to institute the "stir up regimen

Preoperative Status of the Patient—Patients with respiratory complications i e the common cold bronchiectasis etc are often predisposed to lung complications of this nature

Sedation—Too heavy postoperative sedation for pain is dangerous particularly in the patient with pre existing lung diseases Not only do the opiates decrease the rate of respirations and diminish the cough reflex but they also slow the action of the cilia in the bronchi and trachea so that secretions are not moved toward the larynx

Pain from Surgery—This perhaps is the outstanding offender because it decreases the depth of respiration which in turn results in poor aeration of the alveoli The patient particularly the one with upper abdominal incisions often will not take deep breaths i e he "splints respirations so as not to aggravate or bring on pain

Stir up Regimen—In most institutions no effort to make the patient follow a "stir up regimen" i e cough move from side to side or take deep breaths is made by the nursing or house staff unless specific instructions are written that this be carried out at specified times and the results be charted

SIGNS AND SYMPTOMS

Physiological Findings—Increase in temperature, rate of respiration and tachycardia are usually the first signs of respiratory distress Cough and hypoxia rapidly follow However occasionally a cough may be the first warning sign and if while making post operative rounds the patient is noted to cough every 20 to 45 seconds some lung complication should be suspected In these cases the temperature and pulse should be checked and auscultation of the chest should be performed Even if physical examination is negative the patient should be carefully observed for the development of the signs and symptoms of respiratory complications during the next 24 hours

Roentgen Studies—X ray evidence of the disease is usually present and confirms the physical findings If x ray is to be used for diagnosis a 6 foot film should be taken since bedside x rays are usually of little value and are often misleading (see Figure 16 page 61)

PROPHYLAXIS

Steps to prevent pneumonia and atelectasis should be instituted prior to surgery and during anesthesia as well as in the postoperative

period All too often the physician does not think of the prevention of lung complications until the patient is plagued with one and this may be too late

Prophylactic Treatment Prior to Anesthesia—In patients with pre existing lung disease preoperative antibiotic therapy postural drainage and bronchoscopy are indicated to minimize the accumulation of secretions If noninfected bronchorrhea from silicosis etc is present a course of adrenal extract therapy (Cortisone etc) 2 to 3 weeks prior to elective surgery will often reduce the bronchorrhea to a minimum However if Cortisone is used in this fashion it must be continued during surgery and the first 3 to 5 days postoperatively Otherwise severe complications or death may result [1]

The preoperative medication should be carefully selected and given in the correct dosage so as not to depress respiration Waters [6] has shown that if morphine and a belladonna drug are administered in the ratio of 1 25 maximum sedation will ensue with minimal respiratory depression

The stomach should be emptied of its contents prior to surgery This may necessitate only the restriction of oral intake 12 hours before surgery or it may necessitate the use of a Levin or Ewald tube This procedure may preclude vomiting or silent regurgitation of stomach content during the anesthetic procedure the operation and the postoperative period

Prophylactic Treatment During Anesthesia—In the anesthetized patient gross vomiting or silent regurgitation of stomach contents is particularly dangerous [11] If the emesis should be inhaled the hydrochloric acid may destroy the epithelium of the alveoli and bring about a chemical pneumonia Therefore if a patient vomits or is suspected of silent regurgitation every attempt should be made to remove the vomitus before it reaches the alveoli for they suffer the greatest damage from the acid Some anesthesiologists feel that blind suction through a large endotracheal tube is as effective as bronchoscopy We do not share this opinion because as stated in Chapter 18 page 168 it is our belief that "a peek is worth two finesses If a patient aspirates the only certain method of being sure that stomach contents are not left in the trachea and bronchi is by means of the bronchoscope

Prophylaxis in Postoperative Period—Postoperatively the following care to prevent lung complications should be instituted as indicated by the patient s condition

Stir up Regimen—This includes (1) turning the patient from side to side (2) having the patient when awake take 5 to 10 deep breaths every hour for the first 48 hours (3) urging him to cough to remove mucus and secretions from the lung and (4) if necessary having him sit on the edge of the bed and making him cough as he is pounded gently on the back to loosen plugs Since improperly applied abdominal binders may greatly restrict respiratory efforts the physician should check to see that they are applied properly and are not restricting respirations

Intercostal Blocks—These are of particular value if the patient refuses to cough because of pain following upper abdominal operations More often than not once the pain is relieved these patients will be most cooperative in carrying out the "stir up regimen" [7 9]

Tracheal Suction—If the patient refuses to cough even after an intercostal block then he may be made to cough by passing a urethral catheter through the nose into the trachea Just sucking out the pharynx is of little help in these cases but it is all that may usually be expected if the task is left to a nurse rather than performed by a physician familiar with this technique

Bronchoscopy—If in spite of the above therapy the patient develops atelectasis or regurgitates fluid from his stomach into his trachea and cannot expel it because he is too weak then bronchoscopy must be performed

Antibiotic Therapy—If any aspiration of gastric contents occurs if atelectasis is present or if a pneumonia unheralded by these situations develops appropriate antibiotic therapy i e penicillin Terramycin etc should be started at once in an effort to pre

vent pneumonia It must be remembered that administration of the antibiotics following aspiration of the stomach contents does not directly help the healing of the alveolar epithelium damaged by the hydrochloric acid in the digestive juices but it does help to prevent secondary infection

TREATMENT

Active treatment of atelectasis or bronchopneumonia is the same as prophylactic treatment in the postoperative period (see above) In addition, oxygen by nasal catheter bag, and mask or by tent may be indicated Oxygen should not be saved as a last resort for the dying but used rather as a valuable therapeutic agent for the living[10]

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CHAPTER 31

Hiccough and Paralytic Ileus

Hiccough —The relationship of hiccoughs to a regional block procedure either during surgery or following it is difficult to evaluate Most of the cases of hiccoughs which have occurred at the Mason Clinic during surgery have clearly been related to traction on the stomach ileum gall bladder or spleen and have ceased shortly after the pull on these organs was suspended

If hiccoughs are troublesome to the surgeon and traction on the viscera cannot be suspended it has been our finding that the only sure way of stopping the hiccoughs has been the administration of cyclopropane until apnea has occurred and then performing controlled respirations for 5 minutes When the patient is allowed to regain consciousness the hiccoughs have usually ceased The use of d desoxyephedrine in 10 mg doses intravenously has been suggested as a means of relieving hiccough We have tried this in 5 patients and were only able to relieve the hiccoughs in 1

On occasion Pentothal anesthesia has been effective in stopping this complication but its effects have not been as consistent as the use of cyclopropane

Paralytic Ileus —Paralytic (adynamic) ileus may occur following any type of anesthetic procedure and it may occur in cases where no anesthesia is administered Paralytic ileus is usually a direct sequel of peritoneal irritation Best and Taylor [1] state The most common cause of paralytic ileus is peritonitis it may also result from severe intestinal injury or undue handling and exposure of the bowel during abdominal operations It has been our finding at the Mason Clinic that the most serious and persistent cases of paralytic ileus have occurred following retroperitoneal operations particularly kidney explorations

While paralytic ileus may occur following a regional block it is highly unlikely that the block initiates it *It should be noted that one* of the treatments of paralytic ileus is spinal epidural or celiac plexus block Any one of these blocks paralyzes the abdominal portion of the sympathetic nervous system leaving the parasympathetic nervous system unopposed and as a result the bowel contracts and expels the gas [2, 3, 4]

Paralytic ileus may also occur in patients who are receiving Banthine (methantheline) therapy These cases may come to surgery for a condition other than that for which the Banthine is being administered If they develop paralytic ileus following surgery the ileus may be unrelated to either the surgery or the anesthesia [6]

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Complications of Specific Regional Nerve Blocks

THE CHAPTERS in this book are arranged to deal with the specific complications of regional nerve blocks in general. No effort is made to list a specific regional nerve block procedure, i.e., brachial plexus block, stellate ganglion block, etc., and then to enumerate its complications.

Therefore the Appendix names the commonly used regional block procedures, listing the complications which may follow their use, and gives the page or pages where this particular complication is discussed in detail. In this manner the Appendix serves as a supplement to the Index, and for convenience the nerve blocks are placed in alphabetical order.

Abscess formation, cellulitis, slough, bleeding into the tissues from trauma to blood vessels, breaking of a needle, pain following insertion of a needle, swelling at the site of the block from bleeding or the injection of too much anesthetic solution, neuritis, dermatitis, and systemic reactions are all inherent hazards of any regional block procedure. While their repeated listing under each block named may appear repetitious, it seems advisable to fulfill the purpose of this section by a thorough listing.

The infrequency with which complications occur following a regional block procedure usually precludes the possibility that all the complications which may occur following a specific block procedure will have been observed at any one institution regardless of the number of blocks performed. Therefore the list of complications found under each specific regional block procedure represents the complications of that block as compiled from medical literature as well as the experience of the author and his colleagues. Those complications observed by the author and/or his associates at the Mason Clinic are italicized. The italicizing of a term should not be construed to mean that this complication has been observed frequently—in many instances it may have been seen only once or twice.

BRACHIAL PLEXUS BLOCK

Abscess, cellulitis, slough, ulceration, 93, 150
Bleeding, 87
Broken needle, 242
Cardiac failure and death, 7, 69
Cerebral air embolism, 66
Dermatitis, 127
Dislocated shoulder, 112
Hematoma, 69
Hemopneumothorax, 65
Hemoptysis, 61
High or total spinal block, 48
Horner's syndrome (paralysis of cervical sympathetics), 120
Mediastinal emphysema, 62
Neuritis, 112, 114, 119
Overflow onto other nerves, 47, 120
Oxygen want (primary), 42
Pain, 84
Pain simulating that of pneumothorax, 65
Phrenic nerve block, 43
Pleural effusion, 62
Pleural shock, 66
Pneumothorax, 55, 56
Stellate block, 120
Subcutaneous emphysema, 62
Swelling, 89, 94
Systemic reaction to
  local anesthetic agent, 6, 33
  vasoconstrictor drug, 37

CAROTID SINUS NERVE BLOCK

Abscess, cellulitis, slough, ulceration, 93, 150
Bleeding, 87
Broken needle, 242
Cardiac failure and death, 7, 69
Dermatitis, 127
Horner's syndrome (paralysis of cervical sympathetics), 120

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Complications of Specific Regional Nerve Blocks

THE CHAPTERS in this book are arranged to deal with the specific complications of regional nerve blocks in general. No effort is made to list a specific regional nerve block procedure, i.e., brachial plexus block, stellate ganglion block, etc., and then to enumerate its complications.

Therefore the Appendix names the commonly used regional block procedures, listing the complications which may follow their use and gives the page or pages where this particular complication is discussed in detail. In this manner the Appendix serves as a supplement to the Index, and for convenience the nerve blocks are placed in alphabetical order.

Abscess formation, cellulitis, slough, bleeding into the tissues from trauma to blood vessels, breaking of a needle, pain following insertion of a needle, swelling at the site of the block from bleeding or the injection of too much anesthetic solution, neuritis, dermatitis, and systemic reactions are all inherent hazards of any regional block procedure. While their repeated listing under each block named may appear repetitious, it seems advisable to fulfill the purpose of this section by a thorough listing.

The infrequency with which complications occur following a regional block procedure usually precludes the possibility that all the complications which may occur following a specific block procedure will have been observed at any one institution regardless of the number of blocks performed. Therefore, the list of complications found under each specific regional block procedure represents the complications of that block as compiled from medical literature as well as the experience of the author and his colleagues. Those complications observed by the author and/or his associates at the Mason Clinic are italicized. The italicizing of a term should not be construed to mean that this complication has been observed frequently—in many instances it may have been seen only once or twice.

BRACHIAL PLEXUS BLOCK

CAROTID SINUS NERVE BLOCK

EPIDURAL (PERIDURAL) BLOCK (cont)

FACIAL NERVE BLOCK

FEMORAL NERVE BLOCK

FEMORAL NERVE BLOCK (cont)

GASSERIAN (SEMILUNAR) GANGLION BLOCK
(Trigeminal Nerve Block)

GLOSSOPHARYNGEAL NERVE BLOCK

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN CERVICAL AREA

See Stellate Ganglion Block, page 264

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN LUMBAR AREA

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN THORACIC AREA

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN THORACIC AREA (cont.)

PENIS BLOCK

PHRENIC NERVE BLOCK

(Local infiltration of phrenic nerve *per se* as it lies on the anterior scalenus muscle)

PRESACRAL NERVE BLOCK

PUDENDAL NERVE BLOCK

RADIAL NERVE BLOCK AT ELBOW OR WRIST

SCALP BLOCK

SCIATIC NERVE BLOCK

SCIATIC NERVE BLOCK (cont)

SPINAL (SUBARACHNOID) BLOCK

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN CERVICAL AREA

See Stellate Ganglion Block page 264

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN LUMBAR AREA

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN THORACIC AREA

PARAVERTEBRAL SYMPATHETIC GANGLION BLOCK IN THORACIC AREA (cont)

PENIS BLOCK

PHRENIC NERVE BLOCK

(Local infiltration of phrenic nerve per se as it lies on the anterior scalenus muscle)

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